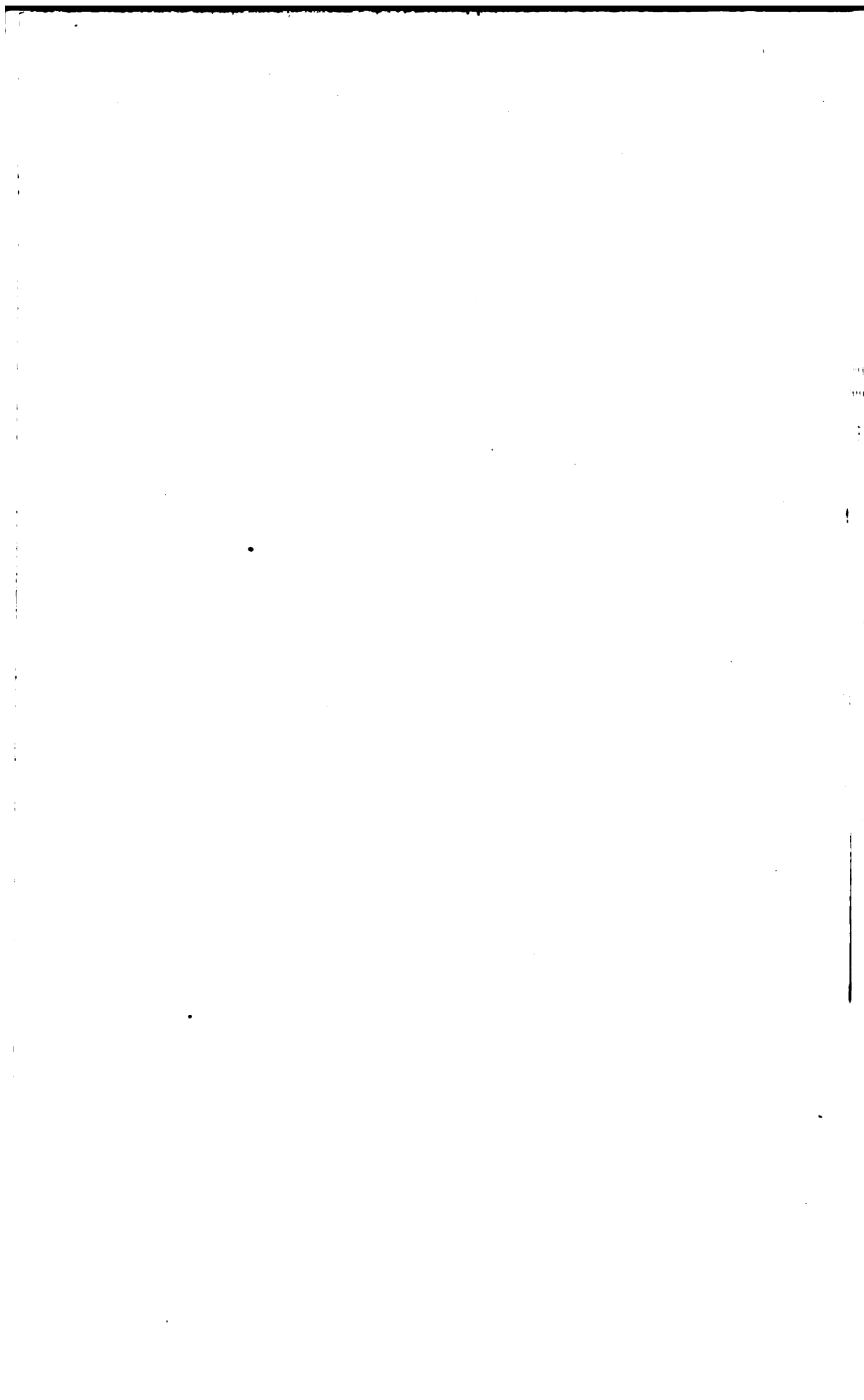


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Review of Neurology & Psychiatry

(Founded by the late Dr Alexander Bruce)

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**REVIEW OF
NEUROLOGY AND PSYCHIATRY**

REVIEW

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(FOUNDED BY THE LATE DR ALEXANDER BRUCE)

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UNIV. OF
CALIFORNIA

Review

of

Neurology and Psychiatry

Original Articles

THE RELATIVE AMOUNTS OF GREY AND WHITE MATTER IN SOME NORMAL AND PATHOLOGICAL BRAINS.

Preliminary Communication.¹

By JOHN CRUICKSHANK, M.D.,

Crichton Royal Institution, Dumfries; Temporary Lieutenant, R.A.M.C.

THE marked complexity of the convolutions of the brain of man, as compared with the lower animals, has suggested to numerous writers that the higher intellectual and other mental faculties characteristic of the human subject are in more or less direct relationship to the amount of grey matter in the brain. Attempts have therefore been made to measure the amount of grey matter in the brains of persons of very different degrees of intelligence and mental development. Owing to the highly complicated nature of the convolutions of the human brain, the method of estimation by direct dissection of the grey matter from the underlying white matter has not been adopted, except in the case of a very few brains, most workers having approached the subject by indirect methods. Some observers have directed their attention to the measurement of the surface area of the grey matter, others to the determination of the absolute amount of

¹ Read at a meeting of the Scottish Division of the Medico-Psychological Association of Great Britain and Ireland, at Edinburgh, on the 17th November 1916.

cortex. Danilewsky, as a result of observations upon the specific gravity of the brain and of the grey and white matter, has calculated that the cortex forms 30 per cent. of the total brain weight. Donaldson, on the other hand, has estimated the grey matter of the hemisphere as forming 50 per cent. of the whole.

In the work of which this paper is a brief summary, an attempt has been made to measure by direct dissection the relative amounts of grey and white matter in a small series of normal brains, and to compare the results with the findings in brains of cases of mental disease which at post-mortem examination exhibited varying degrees of atrophy. The procedure was as follows:—The membranes having been carefully stripped, the pons medulla and cerebellum were removed by cutting through the mid-brain as close to the hemispheres as possible. The hemispheres were then separated by mesial section, and the fluid expressed from the ventricles. One hemisphere was then laid on its mesial surface, and divided into five portions, named, for convenience in reference, the frontal, pre-central, post-central, occipital, and temporal portions. Each portion, which weighed approximately 100 gm., was then cut into slices about one-tenth of an inch in thickness, one slice only being cut at a time, the rest of the piece, along with the other parts of the brain not actually under examination at the time, being kept in a closed vessel in the ice-chest in order to prevent drying and decomposition. Each slice as it was obtained was laid on a glass plate and cut into smaller portions, and by a combination of cutting and scraping with a sharp scalpel the grey matter was separated from the white. It was found that considerable practice was necessary before a reliable separation of the two layers could be made, and the results obtained from the earlier specimens of brain had to be discarded. The work was exceedingly laborious, as, even with the help of an assistant, each of the five portions of the hemisphere took from four to six hours for complete separation. As each slice was completed the separated material was placed under cover. It was found that the fresh unfixed brain gave the best results, as the difference in consistency of the softer grey and firmer white matter in the fresh brain was a very material aid in the separation. The necessity which the use of the unfixed brain imposed of carrying the dissection through in the shortest possible time increased the arduous nature

of the work. The difficulty of separation was greatest at the occipital and frontal poles, owing to the small size of the convolutions and the degree of infolding of the surface. The pre-central and post-central portions were, on the other hand, comparatively easy, the proportion of white matter in these regions being large, and the convolutions much wider. The dissection of the grey matter of the basal nuclei presented the greatest difficulty. The grey matter of the basal nuclei, averaging about 20 gm., was not reckoned as cortical grey matter, and is not included in the following results.

In the tables are given the main results with the series of five normal and eleven pathological brains investigated. The figures represent the amounts and percentages of matter in one hemisphere only. Table I. shows the results when the normal and the pathological brains respectively are arranged according to the amount of grey matter in the members of each group. It will be seen that in the normal brains the weight of grey matter varied from 327 gm. to 253 gm., and the white matter from 237 gm. to 196 gm., the variations in weight corresponding mainly to the size of the brain. The weight of grey matter, expressed as percentage of the total grey and white, varied from 57.9 per cent. to 53 per cent. It is to be noted that, generally speaking, in the normal series the greater the amount of grey matter, the greater was the amount of white matter. In the pathological series the grey matter varied from 289 gm. to 218 gm., and the white matter from 259 to 149 gm. The percentage of grey matter varied from 64 to 52.6 per cent.

Table II. shows the results when the brains, normal and pathological, are arranged in three series or columns, in the order respectively of (A) their amounts of grey matter, (B) their amounts of white matter, and (C) their percentages of grey matter. In column A it is to be noted that the normal brains are distributed at various levels throughout the series, namely, two at the top, one in the middle, and two near the bottom. That is to say, the absolute amount of grey matter in the hemisphere is not a distinctive feature of the normal brains. In column B, however, the normal brains are all in the upper half of the column. The normal brains, in fact, differ from the pathological brains, with one marked exception, in having absolutely greater amounts of white matter. This is expressed differently in column C, in which it is

shown that the pathological brains, owing to the loss of white matter, have a percentage of grey matter, in proportion to the total grey and white, greater than in the case of the normal brains. The pathological brains, with one exception (Mr R.), showed varying degrees of atrophy, and the results of the work have shown that the greater the degree of atrophy of the brain, the greater is the diminution in the amount of white matter. In Mr R.'s case—a voluntary boarder who suffered from melancholia of a few years' duration—the brain was large and apparently healthy, no trace of atrophy being visible. Microscopically this brain was practically indistinguishable from a normal brain, and in the tables it will be noted that it falls among the group of normal brains.

While the figures in the tables give the weights of material actually obtained after separation, it is to be remarked that a certain loss of tissue, particularly of grey matter, occurred during the process of separation. The total loss of matter per hemisphere amounted on the average to about 30 gm., of which it is estimated that about 20 gm. consisted of grey matter.

The conclusions which have been drawn from these results are that the atrophy of the brain, which is so common a feature at autopsy in chronic cases of insanity, is due more to the loss of the underlying white than to the loss of the superficial grey matter, notwithstanding the well-known morbid histological changes in the latter. This relatively greater loss of the white matter of the brain in chronic insanity is quite in keeping with our present knowledge of the neuron, when we remember such facts as the association of the myelination of nerve fibres with the acquisition of higher neural and mental function in the process of development, and the essentially nutritive rôle of the body and nucleus of the nerve cell. Further, the figures which have been obtained for the weight of the grey and the white matter in the different parts of the brain, and which are not published here, have shown that the loss of white matter is greatest in the occipital, temporal, and frontal lobes, and that the white matter of the pre-central and post-central regions suffers to a less degree. In this short communication it has only been possible to give the most striking results. A paper with fuller particulars regarding the age and sex of the patients from whom the pathological material was obtained, the nature and

GREY AND WHITE MATTER

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duration of the mental illness, and other details, will be published later.

TABLE I.—Showing in a Cerebral Hemisphere of each of the Normal and Pathological Brains (1) the amount of Grey Matter, (2) the amount of White Matter, and (3) the percentage of Grey Matter.

Brain.	(1) Grey Matter (Grammes).	(2) White Matter (Grammes).	(3) Grey Matter (Percentage).
Normal. No. 8 -	327	237	57·9
„ „ 7 -	290	227	56·0
„ „ 10 -	275	227	54·7
„ „ 6 -	254	223	53·0
„ „ 9 -	253	196	56·3
Pathological, Mr R.	289	259	52·6
„ Mrs T.	288	205	62·4
„ Mr L.	287	161	64·0
„ Miss T.	286	173	58·2
„ Mrs D.	282	163	63·4
„ Miss B.	268	173	60·7
„ Mrs B.	262	149	63·7
„ Mr C.	261	173	61·5
„ Mrs A.	261	174	59·9
„ Mr F.	235	177	57·1
„ Mrs F.	218	163	57·2

TABLE II.—Showing the Brains, Normal and Pathological, arranged in three series, in the order respectively of their (A) amounts of Grey Matter, (B) amounts of White Matter, and (C) percentages of Grey Matter.

A		B		C	
Brain.	Grey Matter (Gm.).	Brain.	White Matter (Gm.).	Brain.	Grey Matter Per Cent.
Normal—No. 8 -	327	Mr R. -	259	Mr L. -	64·0
„ No. 7 -	290	Normal—No. 8	237	Mrs B. -	63·7
Pathological—Mr R. -	289	„ No. 7	227	Mrs D. -	63·4
„ Mrs T. -	288	„ No. 10	227	Mrs T. -	62·4
„ Mr L. -	287	„ No. 6	223	Mr C. -	61·5
„ Miss T. -	286	Mrs T. -	205	Miss B. -	60·7
„ Mrs D. -	282	Normal—No. 9	196	Mrs A. -	59·9
Normal—No. 10 -	275	Mr F. -	177	Miss T. -	58·2
Pathological—Miss B. -	268	Mrs A. -	174	Normal—No. 8	57·9
„ Mrs B. -	262	Miss T. -	173	Mrs F. -	57·2
„ Mr C. -	261	Mrs B. -	173	Mr F. -	57·1
„ Mrs A. -	261	Mr C. -	173	Normal—No. 9	56·3
Normal—No. 6 -	254	Mrs D. -	163	„ No. 7	56·0
„ No. 9 -	253	Mrs F. -	163	„ No. 10	54·7
Pathological—Mr F. -	235	Mr L. -	161	„ No. 6	53·0
„ Mrs F. -	218	Mrs B. -	149	Mr R. -	52·6

THE WATER CONTENT OF SOME NORMAL AND PATHOLOGICAL BRAINS.**Preliminary Communication.¹**

By JOHN CRUICKSHANK, M.D.,

Crichton Royal Institution, Dumfries ; Temporary Lieutenant, R.A.M.C.

IN the preceding paper it has been shown that the general or local atrophy of the brain occurring in chronic insanity is very largely due to loss of white matter. It seemed to be of interest to determine what chemical changes accompanied this shrinkage in the size of the brain. As a preliminary to an examination of the complex substances of which brain matter is composed, it was necessary to investigate the proportion of water to solids, as the figures obtained in this way are of the highest importance in regard to the interpretation of the results obtained by chemical methods. The examination of the various portions of brain tissue for the amount of water was therefore proceeded with as a routine measure. The whole of the grey or of the white matter, as the case might be, from each of the five portions, obtained as described in the preceding paper, was spread on glass plates in as thin a layer as possible and carefully weighed. The plates were then placed in a Hearson electric drying oven, the temperature of which was maintained at about 90° C. A current of dry hot air was passed into the oven from a fan attached to a small motor. After fifteen to twenty hours' exposure the plates were removed from the oven, and the solid material which remained was carefully and completely scraped off. It was then allowed to cool to room temperature and weighed. The material was returned to the oven for some hours, again removed and allowed to cool. This procedure was continued until the material attained a constant weight at room temperature. The percentage of water in each sample was then calculated.

The following tables show that the grey matter contains roughly 10 per cent. more water than the white, not only in the case of normal brains, as has been observed by others, but also in the pathological and atrophic brains. From Table I. it will be seen that, in the series of five normal and sixteen pathological brains examined, there is considerable variation in the amount of water, not only in different brains, but also, though to lesser extent,

¹ Read at a meeting of the Scottish Division of the Medico-Psychological Association of Great Britain and Ireland, at Edinburgh, on the 17th November 1916.

in different portions of the same brain. In the case of the pathological brains, the amount of water is, in the majority of cases, greater than in the corresponding portions of the normal brains. This is most evident in the case of brains 19, 21, 33, and 37. In Table II. are given the average percentages of water in the grey and the white matter of the different portions of the cerebral hemispheres of the brains in the two series. The pathological series shows the greater water content in both grey and white matter in all the portions. Further, the increase in the amount of water in the pathological series is greatest in the white matter of the frontal and occipital regions. The grey matter of these regions also shows a marked increase in water.

It was found that the more marked the degree of atrophy of the brain, the greater was the amount of water in the brain tissue. The amount of water was always increased in brains which showed marked atheroma of the basal or other arteries.

TABLE I.

Showing the Percentages of Water in the White and the Grey Matter of the different portions of the Cerebral Hemispheres of Normal and of Pathological Brains.

Brain.	Frontal.		Pre-central		Post-central.		Occipital.		Temporal.	
	White	Grey	White	Grey	White	Grey	White	Grey	White	Grey
Normal, 25 - -	73.1	82.8	71.6	81.4	71.2	79.6	70.8	81.7	72.9	81.1
„ 26 - -	70.7	81.5	70.2	81.5	70.5	81.5	69.8	80.7	73.0	82.3
„ 28 - -	72.0	81.4	71.4	81.1	70.8	81.1	71.3	80.4	73.5	81.6
„ 30 - -	73.0	83.2	72.1	83.0	71.6	82.3	72.8	81.7	75.9	83.5
„ 34 - -	73.5	84.6	75.2	83.7	72.4	83.3	72.3	82.5	74.5	83.6
Pathological, 16 -	72.1	82.4	74.5	81.5	72.9	80.9
„ 17 -	74.9	82.7	73.4	81.9	72.9	81.8	76.2	81.9	74.0	83.4
„ 19 -	77.7	83.4	76.0	82.1	75.4	83.4	75.1	82.7	78.5	83.6
„ 20 -	71.2	82.6	70.0	81.9	71.9	79.9
„ 21 -	77.5	85.8	75.9	85.6	77.5	85.6
„ 22 -	72.1	81.0	70.0	82.5	71.0	80.2	72.3	81.9	71.4	78.9
„ 24 -	73.4	83.2	72.1	83.0	70.0	80.4	71.7	80.8	73.2	80.4
„ 27 -	74.2	83.9	71.8	82.4	73.7	82.1	73.0	82.5	75.0	84.1
„ 29 -	71.8	82.7	71.4	82.6	69.9	81.4	71.7	82.1	71.2	82.5
„ 31 -	69.4	82.2	69.0	81.7	70.5	80.5	67.5	80.2	71.5	81.2
„ 32 -	74.1	84.9	76.1	86.3	73.4	85.3	73.5	84.8	71.5	87.1
„ 33 -	79.2	85.6	76.9	82.0	75.2	82.4	76.5	82.8	80.0	83.8
„ 35 -	75.0	84.2	73.6	84.7	73.5	84.2	73.2	82.9	76.5	83.3
„ 36 -	75.9	83.6	75.2	81.5	74.4	80.9	74.2	81.8	74.9	83.6
„ 37 -	76.1	86.2	75.2	83.8	75.0	83.2	71.7	85.2	76.0	85.0
„ 38 -	74.9	86.8	71.2	85.1	69.2	83.7	72.9	83.6	75.0	84.3

TABLE II.

Showing the Average Percentages of Water in the White and the Grey Matter of the different portions of the Cerebral Hemispheres of Normal and of Pathological Brains.

Brain.	White Matter.					Grey Matter.				
	Frontal	Pre-central	Post-central	Occipital	Temporal	Frontal	Pre-central	Post-central	Occipital	Temporal
Normal - -	72.4	72.5	71.4	71.4	73.9	82.7	82.7	81.5	81.4	82.4
Pathological -	74.3	73.2	72.6	73.2	74.5	83.8	83.0	82.2	82.4	83.1
Difference -	1.9	0.7	1.2	1.8	0.6	1.1	0.3	0.7	1.0	0.7

Abstracts

ANATOMY.

- HISTOLOGICAL STUDIES ON THE LOCALISATION OF CEREBRAL FUNCTION.—THE BRAIN OF THE GORILLA.** ALFRED W. CAMPBELL, *Reports from Path. Lab. of Lunacy Department, New South Wales Government*, 1916, iii., pp. 19-35 (5 figs.).

THE brain examined was the right cerebral hemisphere of a baby gorilla, and this paper is to be regarded as an addendum to the writer's well-known monograph on the localisation of cerebral function (*v. Review*, 1906, iv., p. 308). He recognises in this brain all the areas previously found in the cerebral cortex of man, the chimpanzee, and the orang-outang, and in addition describes two new areas not previously recognised, viz., the "subtemporal" and the "extra-olfactory."

The "subtemporal" area is confined to the lower surface of the lobe, and covers the whole of the third and a considerable portion of the fourth temporal gyrus (gyrus fusiformis). It has the same inferior boundaries as the original area, except that it is separated from the olfactory area (lobus pyriformis) by a zone of cortex, now revealed also for the first time in the anthropoid brain, and termed the "extra-rhinc" or "extra-olfactory" area. It is suggested that this latter probably represents an olfactivo-psychic area, and thus brings our conception of the olfactory area, as possessing a sensory and a psychic portion, into line with other sensory realms.

A. NINIAN BRUCE.

- THE RELATION OF BRAIN TO SKULL: WITH SPECIAL
(2) REFERENCE TO THE AUSTRALIAN ABORIGINAL.** J.
FROUDE FLASHMAN, *Reports from Path. Lab. of Lunacy Department,
New South Wales Government*, 1916, iii., pp. 1-15 (8 plates).

THE object here attempted was to discover if any evidence could be obtained by investigating brain to skull relationship of any indication of a common line of phylogenetic development passing from the cynocephalus through the orang and the aboriginal to the European. Serial horizontal sections of the head were made, and the corpus callosum was used as a suitable soft internal structure from which to measure the relation of its longest axis to (1) the basi-cranial axis, (2) the plane of the foramen magnum, and (3) the general direction of the spinal canal being ascertained. Except in the case of the relation of the corpus callosum to the general line of the cervical vertebræ, no definite gradation was found. This is due to the fact that the measuring points are too variable. The brain in the skull possesses an independent capacity for rearrangement of the functions of its various parts, and can expand one part and contract others in reponse to the demands of the environment, which may be only local and temporary, but which prohibit the view of a definite progressive relationship between parts of the soft active cerebrum and the hard exterior cranium.

A. NINIAN BRUCE.

- THE HISTORY OF THE EYE MUSCLES.** H. V. NEAL, *Proc.
(3) Amer. Assoc. Anatomists*, Dec. 1916; *Anat. Record*, 1917, xi., Jan.,
p. 391.

NEAL attempts to demonstrate on the basis of embryological evidence the exact homology of the first three permanent myotomes of *Amphioxus*, *Petromyzon*, and *Squalus*, and to describe the more important stages in the phylogenesis of the eye muscles. He presents evidence for the first time to support the assertion of Dohrn (1904), and of Neal (1907), that the second as well as the third myotome participates in the formation of the external rectus muscle. Thus he amends the familiar text-book formula for the ontogenesis of the eye muscles as follows:—From the first myotome (the pre-mandibular head-cavity) arise the muscles innervated by the oculomotor nerve. From the second myotome (the mandibular head-cavity) develop the superior oblique muscle, and also the ventro-lateral part of the external rectus. From the third myotome (the hyoid head-cavity) arises the dorso-median portion of the external rectus muscle.

LEONARD J. KIDD.

THE PINEAL REGION IN HUMAN EMBRYOS. JOHN WARREN,
(4) *Proc. Amer. Assoc. Anatomists*, Dec. 1916; *Anat. Record*, 1917,
xi, Jan., p. 428.

WARREN'S object is to call attention to three special features in the development of the pineal region in human embryos.

1. *The Primary Arches in the Roof of the Fore-Brain.*—In an embryo of 10 mm. all these arches are clearly differentiated. A low velum separates a well-marked paraphysal arch from a relatively short and thick-walled post-velar arch. The epiphysal arch is sharply defined, with also rather thick walls, and is succeeded by a relatively long pars intercalaris. This part of the brain roof forms a low arch, in the posterior end of which the posterior commissure can be seen. The commissure seems to appear in this portion of the forebrain before invading the roof of the mid-brain. In an embryo of 15 mm. the arches are more fully developed. "The primary arches at first described by Professor Minot in *Acanthias* can therefore be demonstrated in human embryos, and in addition the pars intercalaris forms an arch of relatively great length as compared with its appearance in lower vertebrates."

2. *Paraphysis.*—The earliest trace was noted as a slight thickening in the paraphysal arch in the embryo of 15 mm., and in two others of 16 mm. The oldest embryo in which any trace of it could be made out was one of 44.3 mm. "The paraphysis therefore does exist in certain human embryos, but it is a rudimentary and inconstant structure. A short but well-defined paraphysal arch could be followed in all the embryos studied." Warren gives other particulars.

3. *Post-Velar Arch.*—A complicated prolongation of the anterior end of this arch just behind the velum forms a striking feature in many embryos. The outgrowth appears either as a median projection or as a bilateral formation on either side of the median line, coming into intimate relation with the vessels over the brain roof. As the outgrowth becomes more complicated, tubules are given off in a rather bewildering manner, which may become detached and appear as blind vesicles buried in the midst of this tubular formation. The paraphysis is more or less covered by this projection, which overhangs to a large extent the paraphysal arch. The formation begins in 23 mm. embryos, and is seen up to the 44 mm. stage.

LEONARD J. KIDD.

THE DEVELOPMENT OF THE HYPOPHYSIS IN REPTILES.
(5) E. A. BAUMGARTNER, *Journ. of Morphol.*, 1916, xxviii., Dec., pp.
209-286 (68 figs.).

THE material used for this study consists of embryos and adults of various turtles, lizards, and snakes, and of the Mississippi

alligator. Conclusions:—"The epithelial portion of the hypophysis develops as a single *anlage* in turtles, lizards, and snakes, and probably in alligators. In the development of the evaginations of the hypophysis, Rathke's pouch appears first, then the two lateral buds, and finally the anterior bud.

"The lateral buds in turtles give rise to the part termed by Tilney 'pars tuberalis'"—(*v. Review*, 1914, xii., p. 200)—"and to a thin cortical zone around the middle of the anterior lobe; in alligators to the pars tuberalis, and two bands encircling the anterior lobe; in lizards they appear to persist as isolated masses or to disappear; while in snakes they completely disappear. The cortical zone or bands present in turtles and alligators have not been described in other vertebrates except in pigs.

"The tip of Rathke's pouch gives rise to the pars infundibularis (Tilney), or pars intermedia of the adult. The remainder of Rathke's pouch and the early anterior bud give rise to the adult anterior lobe, except for the thin cortex or band around it, in turtles and alligators.

"The three parts of the adult hypophysis are distinct ontogenetically and histologically.

"The pars infundibularis or pars intermedia have a laminar arrangement of columnar clear-staining cells. The parts derived from the lateral buds are arranged in columns (or sometimes acini) of clear-staining polyhedral cells. The anterior lobe proper is formed of columns or acini, with clear-staining and darkly-staining cells which may be acidophilic or basophilic. In general, the pars intermedia and the parts derived from the lateral buds may be considered the chromophobic, and the anterior lobe the chromophilic part."

Baumgartner, in discussing the development of the lateral lobes in alligators, has the following interesting passage:—"During development they extend anteriorly and dorsally. In an adult specimen 47.5 cm. long the distal portions of the lateral buds are not continuous across the median line as in adult turtles, while in an adult 150 cm. long the distal portions are fused, and form a single tongue-like pars tuberalis. It would seem, therefore, that there is some growth in the hypophysis, or at least of this part, during adult life. Increase of glandular growth during adult life in *Acanthias* has also been noted" (by Baumgartner, *Journ. of Morphol.*, 1915, xxvi.).

LEONARD J. KIDD.

PHYSIOLOGY.

- THE PHYSIOLOGICAL EFFECTS OF PERIPHERAL SYMPATHECTOMY (LOCAL THERMIC REACTION AND HYPERTENSION).** (Des effets physiologiques de la sympathectomie périphérique (reaction thermique et hypertension locales).) R. LERICHE and J. HEITZ, *Compt. Rend. Soc. de Biol.*, 1917, lxxx., Jan. 20, p. 66.

ABLATION of the peri-arterial sympathetic plexus is followed almost constantly by a vaso-dilator reaction. The writers have practised this operation on the brachial artery in eight cases, and on the femoral artery in one case. Operations were done for a causalgia of the median nerve in one case, and for reflex paretic or spasmodic phenomena of the Babinski-Froment type in the eight other cases. The immediate effect of this operation is a great contraction of the artery, a purely local phenomenon, with loss of pulse and coldness of the periphery. After sometimes only six or seven hours, but commonly twelve or fifteen, rarely not till thirty-six hours after operation, there is vaso-dilatation, with elevation of temperature of the limb: once it was more than 2° higher than on the sound side on the day after operation. The previous coldness may give place to a severe burning sensation. This thermic reaction may vary in degree and duration, but has never been absent. There is also a rise of arterial pressure in the operated limb, and an increased amplitude of the oscillations by Pachon's instrument. These changes are temporary; the local rise of pressure disappears about the fifteenth day; and also the hyperthermia and increased amplitude of Pachon oscillations, though they may occasionally be found for the next ten or fifteen days. The writers refer to the finding by Claude Bernard (1876), that section of the peripheral sympathetic filaments of the horse gave an intense vaso-dilatation.

LEONARD J. KIDD.

PROGRESSIVE MOVEMENTS IN DECEREBRATE KITTENS.

- (7) LEWIS H. WEED, *Proc. Amer. Assoc. Anatomists*, Dec. 1916; *Anat. Record*, 1917, xi., Jan., p. 429.

IN a series of forty kittens subjected to decerebration, different reactions of a rhythmic character were obtained. In all an essentially similar ablation was performed, with removal of cerebral hemispheres and basal ganglia. The brain-stem was cut through just anterior to the superior corpora quadrigemina, sloping forward in the line of the bony tentorium. The kittens

continued to breathe spontaneously, and as soon as the anaesthesia had passed away showed active reflexes and rhythmic progressive movements. Differing from the reactions of adult animals, these decerebrate kittens did not show an invariable extensor rigidity. All, however, gave typical scratch reflexes; they responded to both dorsal and ventral excitation, and reacted to trauma to the tail. In general they were much more active than are the customary adult preparations. A description of the groups into which they can be divided follows. The kittens which showed the phenomenon of prolonged progression varied in age from one hour to sixteen days. For further particulars the paper should be consulted.

LEONARD J. KIDD.

**THE EFFECT OF ROTATION AND OF UNILATERAL REMOVAL
(8) OF THE OTIC LABYRINTH ON THE EQUILIBRIUM AND
OCULAR REACTIONS IN KITTENS.** A. L. PRINCE, *Amer.
Journ. of Physiol.*, 1917, xlii., Jan. 1, p. 308.

THE experiments were performed on kittens ranging in age from six days to seven weeks; the labyrinth was removed by the method of Wilson and Pike (*Phil. Trans. Roy. Soc.*, 1912, B., cciii., p. 127).
Conclusions:—

1. In kittens the general motor reactions which follow rotation and unilateral removal of the otic labyrinth are, up to a certain limit, proportional to the age of the animals. As a whole these reactions seem to progress *pari passu* with the development of the function of equilibration.
2. The ocular reactions, likewise, show progressive changes. The compensatory eye movements following rotation of the head in space are at first of a slow and irregular type, but well defined labyrinthine nystagmus does not appear until about the third week after birth. The rate of post-rotatory nystagmus increases gradually with age.
3. In the younger animals post-rotatory nystagmus is preceded by a latent period.
4. Following unilateral removal of the otic labyrinth there is, up to the end of the second week after birth, deviation of the eyes to the side of the lesion without distinct nystagmus. At three weeks after birth nystagmus, of an intermittent type and of a slow rate, appears. The rate and regularity of the post-operative nystagmus, as in the rotation experiments, increases progressively with the age of the animals.
5. In animals of three to five weeks the post-operative nystagmus disappears relatively early, whereas the deviation of the eyes

to the side of the lesion persists for several days. In older animals the nystagmus and deviation disappear simultaneously.

6. The probable ontophylogenetic importance of these ocular reactions is suggested.

7. The ocular and other phenomena observed following rotation and unilateral removal of the otic labyrinth are explained by a relatively late myelination of the nervous paths concerned.

LEONARD J. KIDD.

THE FUNCTIONAL ANALYSIS OF THE CORTICAL CENTRES

(9) **BY MEANS OF LOCAL CHEMICAL STIMULATION.**

SILVESTRO BAGLIONI, *Quart. Journ. Exp. Physiol.*, 1916, x., p. 169.

PROF. BAGLIONI refers to the paper of Dusser de Barenne (*v. Review*, 1916, xiv., p. 107) upon experimental researches on sensory localisation in the cerebral cortex, and gives a short summary of experiments carried out by himself and his students upon the method of local application of poisons, such as strychnine and phenol, to the nerve-centres which Barenne had apparently overlooked.

A. NINIAN BRUCE.

THE EFFECT OF HYPOPHYSECTOMY ON THE SUBSEQUENT

(10) **GROWTH AND DEVELOPMENT OF THE FROG (*RANA***

***BOYLEI*). P. E. SMITH, *Proc. Amer. Assoc. Anatomists*, Dec. 1916; *Anat. Record*, 1917, xi., Jan., p. 410.**

"IN the operated specimens the hypophyseal rudiment was removed soon after it had commenced to invaginate, *i.e.*, shortly after the closure of the medullary tube. Controls consist of specimens upon which the operation was unsuccessfully attempted, and of unoperated animals. All animals were reared under identical conditions.

"In the hypophysectomised specimens particular attention is called to the non-development of the hind legs; to the pronounced decrease in the size, parenchyma, and colloid of the thyroid gland; and to the profound reduction of the epidermal pigment.

"Only one specimen of the group, in which the hypophyseal rudiment was ablated, developed legs at the normal rate. Sections show that, although the glandular portion of the hypophysis was totally extirpated, yet the thyroid developed normally, and the epidermal pigment was typically reduced."

LEONARD J. KIDD.

PSYCHOLOGY.**THE VERBAL ASSOCIATION TEST IN THE PRIMARY SCHOOL.**

(11) M. EVARD, *Archiv. de Psychol.*, 1916, xvi., July, p. 24.

AN account of a verbal association test applied in two successive years to thirty-two boys, average age for the first experiment being $8\frac{1}{2}$ years. The object was to find how far the year's mental development could be demonstrated by the changes in the responses to the test words. The results are suggestive, but inconclusive. The percentage of intelligent associations increased from 91.70 to 94.65; associations demonstrating analysis or synthesis increased, while co-ordinative and automatic reactions decreased; at the same time there were fewer original responses, while commonplace ones increased in number.

MARGARET DRUMMOND.

MENTAL PROFILES. ED. CLAPARÈDE, *Archiv. de Psychol.*, 1916, (12) xvi., July, p. 70.

THIS article begins with a criticism of the method of obtaining "mental profiles" devised by Rossolimo, of Moscow, in 1911. Certain selected mental powers of an individual, such as observation, verbal memory, &c., are tested, and his achievements indicated on the ordinates of a chart; the points thus obtained are joined, and the resulting curve is known as the mental profile. Claparède points out that Rossolimo's charts are not very informative, because he did not begin by establishing norms. By testing, say, 100 persons taken at random, he himself forms a preliminary scale. The rank of any individual subsequently tested can easily be determined by reference to this scale—not only can we say whether he is above or below average; we can assign to him a definite percentage mark.

A striking illustration of the use of such mental profiles in legal questions is given. A young man had been knocked down by a motor, and his skull had been fractured. This was easily cured, but his family maintained that his mental powers were affected, and they were suing for damages. Claparède was asked to find out whether the claim was justifiable, and to what extent there was injury to the mental ability. He choose about twenty suitable mental tests, tried them on twenty or thirty young people about the age of the subject, and in accordance with these results marked out three zones of ability, below average, average, and above average. He then tested the patient, and found that two-thirds

of his results were below average, and none above average. On comparing his achievements with those of lads younger than himself, Claparède concluded that he might be considered to be about two years mentally retarded. The accident had happened just two years before, and at that time the patient had been one of the most brilliant pupils in his class. Hence it seemed fair to maintain that, as the family contended, the accident had given rise to mental injury.

MARGARET DRUMMOND.

PATHOLOGY.

ON SOME PECULIAR COMPENSATORY MANIFESTATIONS IN

- (13) **THE OPTO-STRIATE NUCLEI.** (*Sopra alcune particolari manifestazioni compensative nei nuclei opto-striate.*) G. D'ABUNDO, *Riv. ital. di Neurop., Psichiatr. ed Elettrotet.*, 1916, ix., p. 343.

THE brain of a hemiplegic idiot who died at the age of 23 showed atrophy of the right hemisphere with absence of the greater part of the frontal lobe, especially in the upper part; atrophy of the corresponding optic thalamus (especially of the antero-internal nuclei), together with enlargement of the caudate and lenticular nuclei (especially of the putamen) and also of part of the anti-murus. D'Abundo had obtained similar experimental results on newborn kittens. After destruction of an optic thalamus through the corpus callosum he had found a compensatory enlargement of part of the corresponding caudate and lenticular nuclei while the cerebral hemisphere was hypotrophic (*cf. Review*, 1913, xi., p. 218).

J. D. ROLLESTON.

HETEROTOPIA OF THE SPINAL CORD. OLIVER LATHAM and J.

- (14) FROUDE FLASHMAN, *Reports from Path. Lab. of Lunacy Department, New South Wales Government*, 1916, iii., pp 53-66 (36 figs.).

THIS abnormality was discovered accidentally while examining the spinal cord of a man, aged 64, who died from tabes, and who showed apparently no other symptoms during life. A tumour was then found at the region of the eighth dorsal segment. The usual tabetic degeneration of the posterior columns was present; the other myelinated tracts appeared quite normal. The tumour consisted of a blindly ending excrescence from the spinal cord containing both grey and white matter, the grey matter being continuous with the grey matter of the anterior cornua. It extended upwards as far as the sixth dorsal segment.

At the level of the fourth dorsal segment it was noticed that a large number of fibres, originating near the tip of the right dorsal horn, crossed the dorso-median septum and the left posterior horn of grey matter to ascend in the attenuated left lateral column. "This appears to be a very definite confirmation of the fact that dorsal root fibres cross and ascend directly to the mesencephalon (at least) without the intervention of any cells in the dorsal horns."

A. NINIAN BRUCE.

CORD SHOWING DEVELOPMENTAL ABNORMALITIES, WITH
(15) **PRIMARY DEGENERATION OF NEURONES IN A CASE**
OF MALIGNANT CERVICAL GLANDS. OLIVER LATHAM,
Reports from Path. Lab. of Lunacy Department, New South Wales
Government, 1916, iii., pp. 180-191 (3 plates).

A WOMAN, aged 48, died on re-admission to hospital some time after she had undergone an operation for malignant glands in the neck. Fourteen days before death she complained of pain in the right side of the neck, three days before death of pains in both arms, one day before death paralysis of both arms developed, and nearly complete paralysis of both legs. Babinski's reflex was present on the left side, undetermined on the right side, the knee jerks were normal, the sphincters unaffected, and no sensory disturbances were noted.

The cord showed numerous excrescences, at first thought to be secondary malignant growths. They were, however, found on section to be protuberances of cord substance covered with pia mater. Some contained medullated fibres running in all directions, with a ground substance like white matter and a few glia fibres. No marked sign of either acute or chronic inflammation or any true tumour formation could be discovered. These congenital malformations had led to alterations in the shape and position of some of the tracts, septas, fissures, and grey matter, and in addition to an altered appearance of some of the neurones, singly and in groups, especially in the cervical region of the cord, where some of the anterior cornual cells presented evidences of moderate degeneration, and where the axis cylinders were swollen, varicosed, and altered in staining properties, with their myelin sheaths dilated, sometimes thinned, and at other times absent. It was not considered that the developmental abnormality was a primary cause of the altered neurones: this was thought to be due to a primary degeneration due to some toxic agent.

A. NINIAN BRUCE.



- HYPERPLASIA OF THE INTERSTITIAL TISSUES OF THE NERVOUS SYSTEM, WITH SYMPTOMS OF MYASTHENIA GRAVIS.** OLIVER LATHAM, *Reports from Path. Lab. of Lunacy Department, New South Wales Government*, 1916, iii., pp. 193-203 (12 figs.).

THE patient was a man, aged 45 years, who suffered from myasthenia gravis, the diagnosis being based upon the following points: easily induced fatigue of muscles recoverable after rest, diplopia, bulbar symptoms, speech defects, myasthenic reaction of muscles, and absence of mental disturbance. The Wassermann reaction was negative.

The pathological changes found in the nervous system included proliferative and degenerative changes in the endothelium and connective tissue cells of the dura and pia mater, with similar interstitial changes in some of the peripheral nerves and posterior spinal nerve roots, and with multiplication of the capsular cells (homologue of the neurilemma) surrounding the posterior spinal ganglion cells. There was a hypertrophy of the neuroglial cells and fibres in the first layer of the cerebral cortex and throughout the nervous system. Irregular pigmentation of the nerve cells in various centres, and absence of proliferative changes in the blood vessels, or of round cells in these or in the pia arachnoid or lymphatics beneath the epineurium, complete the findings. These seem to be dependent on some toxin or deficiency occurring fairly widely in the body fluids, but exercising its greatest effects on the interstitial tissues of the nervous system, hardly explaining, but possibly hastening, the "myasthenic" symptoms.

A. NINIAN BRUCE.

- A CONTRIBUTION TO THE STUDY OF THE ÆTIOLOGY OF DISSEMINATED SCLEROSIS.** J. FROUDE FLASHMAN and OLIVER LATHAM, *Reports from Path. Lab. of Lunacy Department, New South Wales Government*, 1916, iii., pp. 37-51 (2 plates).

A CASE of disseminated sclerosis in a girl, aged 19 years, with prominent spinal symptoms. A definite localised sclerosis of the spinal cord was found, extending for some distance above and below the fifth dorsal segment, at which level the gliosis was so complete as to give rise to the appearance of a complete transverse lesion.

The authors discard the views that the disease is due to a toxæmia, an infection, or the result of a vascular condition. They observed, however, that the epithelium lining the central canal was well defined, and proliferating freely into the surrounding grey matter at the corners. They suggest that these cells, as they sink deeper into the tissues, acquire the characters of rudimentary

neuroglial cells, and thus produce a localised mass of this tissue. They admit, however, that the neuroglia cells in all parts of the central nervous system are capable of taking on a growth far in excess of anything demanded for the support of a neoplastic theory of the kind here suggested, as shown by the occurrence of glio-sarcomata, &c.

A. NINIAN BRUCE.

A CONTRIBUTION TO THE PATHOLOGY OF CHOROIDAL
(18) **MELANOMATA.** R. FOSTER MOORE, *Brit. Journ. Ophthalmol.*, 1916, i, Jan., pp. 26-30 (2 figs.).

MELANOMATA of the choroid are of uncommon occurrence and are usually discovered by accident. This case was that of a man, aged 53, who complained of failing sight, and was found to have well-marked papilloedema. A diagnosis of cerebral neoplasm was made, and a decompression operation was performed. The oedema subsided and the choroidal tumour became evident.

At the autopsy a tumour was found at the left Sylvian point, compressing the frontal, temporo-sphenoidal, and parietal lobes. It could be shelled out from the depression, and measured three inches by two. The choroidal melanoma was composed largely of broad spindle-shaped cells with uneven distribution of pigment. It was probably benign and congenital in origin.

A. NINIAN BRUCE.

TOXIC LESIONS OF THE ADRENAL GLAND. G. S. GRAHAM,
(19) *Journ. Med. Research*, 1916, xxxiv., May, p. 241 (2 plates).

NECROTIC lesions of the adrenal cortex are readily produced in the guinea-pig by the inhalation of chloroform. Similar but less constant and less extensive lesions follow the subcutaneous administration of phenol. Repeated injections of phenol failed to produce any lesions of a chronic type.

Necrosis of the cortical parenchyma is frequent also in infectious conditions both in the guinea-pig and in man. Direct repair of the damaged cortex is effected by mitotic division of the surviving parenchymal and interstitial cells.

In adult animals the zona glomerulosa, and particularly the outer portion of the zona fasciculata, appear to constitute a growth centre where regeneration takes place, and whence the new-formed cells are displaced inward to supply the loss at the inner levels. In the child mitosis may occur throughout the whole depth of the cortex.

Cellular proliferation occurs also in the chromaffine cells of the medulla. It is less frequent and less extensive than that found in the cortex. It was present and notably active in a case of fatal "shock."

A. NINIAN BRUCE.

CLINICAL NEUROLOGY.

- THE EFFECT ON THE REFLEXES OF ARTIFICIAL ANÆMIA**
 (20) **PROCURED BY AN ESMARCH'S BANDAGE.** (*Action de l'anémie expérimentale produite par la bande d'Esmarch sur les réflexes.*) OZORIO DE ALMEIDA and ESPOSEL, *Rev. Neurol.*, 1916, pp. 169-173.

AFTER reviewing the work done on this subject, the authors describe their own experiments. They found that an extensor was replaced by a flexor Babinski reaction in the limb of a paraplegic subject compressed by an Esmarch's bandage, even when the stimulation was applied to the skin of the thigh. They draw the conclusion that the modifications produced are due to the action on the motor organs concerned, and not the peripheral sense organs.

O. P. NAPIER PEARN.

- HERPES ZOSTER: ITS OCCASIONAL OCCURRENCE WITH A**
 (21) **GENERALISED ERUPTION AND ITS OCCASIONAL CONNECTION WITH MUSCULAR PARALYSIS: ALSO AN ANALYSIS OF THE LITERATURE OF THE SUBJECT.** F. PARKES WEBER, *International Clinic*, 1916, Series 26, iii., p. 185.

A REVIEW of the literature, including three personal cases already published (*v. Review*, 1916, xiv., p. 386). J. D. ROLLESTON.

- THE SHAPE OF THE VERTEBRAL COLUMN IN DIFFERENT**
 (22) **POSITIONS, ILLUSTRATED BY ROENTGEN PICTURES.** J. F. FISCHER and K. A. KNUDSEN, *Nord. Med. Arkiv.*, 1916, xlix. (Kirurgi), Afd., h. 4, S. 1-19 (29 figs.).

THE movements of the vertebral column that carry the head from a good carriage of the body to a bad one, and *vice versa*, take place above all in the thoracic part, and especially in its central portion. As it is not possible, by asking people to contract the extensors of their backs, to give them an idea of what they are to do, and as the request that they should straighten their backs will only result in a carrying of the upper part of the trunk backward by bending the loin, it is important to realise that by asking them to lift their heads with the chin drawn in, the thoracic part of the vertebral column will be straightened.

The intercostal spaces are increased when the bending of the thoracic part of the vertebral column is diminished. The thoracic part of the vertebral column is relatively movable when it is a question of the increase of the convexity backward from the

normal position, but very slightly movable when it comes to diminishing this convexity, especially in the central portion.

Voluntary bending sideways of the thoracic part of the vertebral column is possible. The ribs are lifted when the arms are carried in extended position backward, which, no doubt, is due to the fact that the pectoral muscles are greatly stretched by this position.

A. NINIAN BRUCE.

CONTRACTURE IN SPASMODIC PARAPLEGIAS. (Sur la contracture dans les paraplégies spasmodique.) NOICA, *Rev. Neurol.*, 1916, Oct., pp. 257-263.

CONTRACTURE as treated in this article is defined as an exaggeration of the muscular tonicity, set up largely by the diffusion of voluntary stimuli, and to a lesser extent by reflex peripheral stimuli coming from the surface of the skin, and more especially from the deeper structures. For contracture to be produced there must be a double lesion of the pyramidal tracts, and the retention of a relatively large amount of voluntary movement in the limbs of the paraplegics.

The author narrates experiments showing that this contracture becomes more marked on repeated and rapid voluntary movement, and that it appears in one limb when voluntary effort is made with another. Coincident with the latter, he notes an increase of reflex excitability, and attributes the results of "reinforcement" to the effort rather than to the distraction of the patient's attention.

A diagram is given showing how the degree of reflex excitability increases in proportion to the loss of voluntary movement, reaching its maximum in the gravest cases, while contracture is most marked in those of moderate severity.

O. P. NAPIER PEARN.

CARDIAC CRISES IN TABES DORSALIS. REPORT OF A CASE
(24) **WITH SUDDEN DEATH.** M. F. LAUTMAN, *Med. Record*, 1916, xc., p. 722.

A MAN, aged 46 years, who had contracted syphilis twenty years previously and had shown signs of tabes for the last two years, and lately had had difficulty in pronouncing his words, developed attacks of pain about the heart in the nature of cardiac crises. There was no autopsy to explain the sudden death which occurred in one of these attacks.

J. D. ROLLESTON.

EARLY DIAGNOSIS OF TABES DORSALIS. WALTER F. SCHALLER, (25) *Journ. Amer. Med. Assoc.*, 1917, lxxviii., Jan. 20, p. 190.

IN tabes the syphilitic posterior leptomeningitis of the cord is in etiologic relationship to the degeneration of the posterior columns of the cord. Every case of such is thus a potential tabetic. This meningeal reaction is evidenced by an increased cell count and increased globulin content. Repeated lumbar puncture may be necessary to establish a positive Wassermann reaction. Syphilitic radiculitis has its characteristic disturbance of sensibility. The Achillis jerk is usually lost before the knee jerk. Anisocoria and irregular pupils are highly suggestive even if they react to light. Diminished hearing is frequent. Cardiovascular, especially aortic, disease is often found.

A. NINIAN BRUCE.

BILATERAL CHARCOT HIPs, OCCURRING SIMULTANEOUSLY. (26) S. J. WOLFERMANN, *Journ. Amer. Med. Assoc.*, 1916, lxxvii., Nov. 25, p. 1590.

A MAN, aged 42 years, who had had syphilis in 1891 with no secondary symptoms, began to suffer from pains in his knees in 1912. These extended up the thighs, and in about three years he had complete loss of use of both legs. He then presented the following symptoms: lightning pains, loss of knee jerks, sphincter weakness, Argyll-Robertson pupils, and the X-rays showed complete loss of the heads and necks of both femora. Some recovery followed anti-syphilitic treatment.

A. NINIAN BRUCE.

RECENT EPIDEMIC OUTBREAKS OF ACUTE POLIOMYELITIS. (27) A. GARDNER ROBB, *Brit. Med. Journ.*, 1916, ii., Sept. 2, p. 324.

A GENERAL paper. The first clear and definite reference to an epidemic outbreak occurred in 1881, when Bergenholtz recorded eighteen cases in one district in Norway and Sweden, but it was not until 1887 that Medin's record of forty-three cases in Stockholm appeared. In the British Isles the first report of an epidemic outbreak was made in 1897 by Dr Pasteur, who met with seven cases in one family. A brief account is given of some cases in Ireland.

A. NINIAN BRUCE.

THE ETIOLOGY OF ACUTE EPIDEMIC POLIOMYELITIS. GEORGE (28) MATHERS, *Journ. Infectious Dis.*, 1917, xx., pp. 113-124 (4 plates).

IN a bacteriological examination of fresh material from ten cases of acute poliomyelitis, a peculiar polymorphic streptococcus-like organism has been isolated in nine instances, in seven of which

the growth has been pure. Similar organisms have been demonstrated microscopically in the tissues of the central nervous system of three cases. Cultures of this coccus injected into rabbits have produced paralysis of various groups of muscles, and characteristic lesions in the central nervous system, consisting of hyperemia and oedema of the tissues, with hæmorrhages, round-cell perivascular infiltration, and neurophagocytosis in the spinal cord, especially in the grey substance, similar in every detail to the changes considered characteristic of acute poliomyelitis in man. This micrococcus has been recovered from the lesions in the inoculated rabbits by both cultural and microscopic methods.

The artificial cultivation of the poliomyelitis coccus in an ascites-fluid tissue medium under anaerobic conditions causes changes in the media, which cannot be differentiated from those previously described for cultures of the so-called virus of poliomyelitis. Morphologically, also, this bacterium, when grown on the same media, is similar to the virus, and in stained smears it appears in minute Gram-positive coccus-like bodies arranged in pairs, groups, and chains. These minute forms disappear when the organism is cultivated in other media under aerobic conditions.

The morphological, cultural, and pathogenic characters of the poliomyelitis coccus, thus far determined, indicate that it is an important factor in the disease.

A. NINIAN BRUCE.

THE PRODUCTION OF AN ANTI-POLIOMYELITIC SERUM.

(29) JOHN W. NUZUM, *Journ. Amer. Med. Assoc.*, 1917, lxviii., Jan. 6, pp. 24-27 (1 fig.).

By the process of animal immunization with the organisms isolated from the spinal fluids and the central nervous system of human poliomyelitic patients, an antiserum can be produced. Its therapeutic limitations are as yet unknown. Antiserum prepared in this manner contains agglutinins, opsonins, and complement fixation bodies, as well as exhibiting certain antibactericidal properties. It would seem fair to conclude that with potent polyvalent antiserum prepared in this manner, some definite therapeutic value might be anticipated in human cases of the disease.

A. NINIAN BRUCE.

**PROGRESSIVE SPINAL MUSCULAR ATROPHY (DUCHENNE-
(30) ARAN) FOLLOWING ELECTRIC SHOCK: POSITIVE WAS-
SERMANN REACTION.** F. PARKES WEBER, *Proc. Roy. Soc. Med.*, 1916, x., Dec. (Clin. Sect.), pp. 4-6.

AN electrician, aged 48, presented the appearance of great muscular atrophy of both upper extremities and of the muscles

connected with the shoulder-girdle and thorax. About March 1910 he accidentally received a severe shock "from his left to his right hand" (750 volts direct current). The wasting was noticed sixteen months later. The Wassermann reaction on two occasions was positive.

A. NINIAN BRUCE.

MONOCULAR BLINDNESS FOLLOWING DIFFUSED VIOLENCE
(31) TO THE SKULL: ITS CAUSATION AND TREATMENT.

J. HOGARTH PRINGLE, *Brit. Journ. Surgery*, 1917, iv., pp. 373-385.

Loss of vision of one eye as the result of the application of diffused violence to the skull is a well-known fact; the remarkable point is that in most cases the traumatism is comparatively slight, the patient being not even rendered unconscious. This fact probably led to the old idea that the loss of vision was the result of reflex action through the fifth nerve (Beers). Later it was explained as due to "concussion" of the optic nerve, and still later to injury to the nerve in the way of crushing, or tearing of its fibres at the foramen opticum in consequence of fractures involving the foramen (Berlin and Hölder).

The author has had under his charge 309 cases of undoubted fracture of the skull; of these, 140 recovered and only one complained of loss of vision after the accident, and only the left eye was involved. There had been 136 autopsies, in none of which any direct injury to the optic nerve had ever been found, and fractures of the optic foramen were also found to be uncommon. This being so in cases of gross injury inflicted to the skull, it seems hardly probable that direct damage to the nerve should have occurred in the comparatively slight injuries that have been so often the cause of blindness in the reported cases.

The author believes that the condition is due to intravaginal hæmorrhage from rupture of the vessels of the nerve and its sheath, without any fracture of the orbital walls, optic foramen, or any part of the skull. The hæmorrhage may be derived (1) from the cranial cavity and effused into the subdural space, finding its way from the increased intracranial tension into the dural sheath of one or both optic nerves; (2) from rupture of vessels passing between the nerve and its coverings; and (3) from the central vessels of the retina (rare) which run for a short distance inside the dural sheath before they enter the substance of the nerve. Great tension will be produced within the nerve sheath by such an effusion of blood, especially as the dura mater forming the sheath of the optic nerve is very strong, and is attached along the margin of the optic foramen, and blood effused into the orbital side of the foramen would produce compression of the nerve.

The main point regarding the pupil seems to be the absence of its reaction to direct light and the preservation of the consensual reaction.

It is important that all such cases should have their optic nerve exposed at the earliest possible moment after the blindness is discovered, and any blood found in its sheath evacuated.

A. NINIAN BRUCE.

THE WIRE GAUZE BRAIN DRAIN. H. P. MOSHER, *Surgery Gynecology and Obstetrics*, 1916, xxiii., Dec., p. 749.

THIS drain is a metal tube closely studded with perforations. In use it is kept filled with glycerine, and the gauze over it also is saturated with glycerine. The object of this is to soften the necrotic brain tissue and to keep the pus liquid, so that both the cast-off brain substance and the pus can make their way into the tube. The drain may be easily cut from a piece of copper gauze, and may be bent as required.

A. NINIAN BRUCE.

CONTRIBUTION TO THE STUDY OF HYPERTROPHIC PACHY-
(33) **MENINGITIS.** (Contribution a l'etude de la pachymeningite hypertrophique.) G. MARINESCO, *Rev. Neurol.*, 1916, Oct., pp. 233-253.

UNDER this heading the author gives full reports of two cases including the post-mortem histology. He follows the teaching of Charcot and Joffroy in dividing the course of the malady into three stages, namely: a neuralgic stage, the pain being due to the passage of the sensory nerves through the inflamed meninges; a paralytic stage with atrophic paralysis of the upper limb; a stage characterised by a spastic paralysis of the lower limb due to the invasion of the cord by the inflammatory process and consequent secondary degeneration.

The principal phenomena to which the author draws attention in the cases cited are: (1) The so-called "Preacher's Hand" which he regards as not pathognomonic of hypertrophic meningitis but more properly belonging to the domain of syringomyelia. (2) The phenomena of medullary automatism concerning which he quotes the opinion of P. Marie and Foix, considering them as an automatic attempt at the movements of walking. (3) The formation of cavities in the cord, which he thinks incidental to an attempt at the re-establishment of the circulation of the cerebro-spinal fluid, following on the interference with its normal course by the pressure of the thickened membranes.

O. P. NAPIER PEARN.

SPORADIC MENINGITIS IN CHILDREN. P. G. HURFORD,
(34) *Pediatrics*, 1916, xxviii., Dec., pp. 541-547.

FORTY-FIVE cases observed altogether. Of these, twenty-five were tubercular, ten due to the meningococcus, four to the streptococcus, three to the pneumococcus, two to the staphylococcus, and one to the influenzal bacillus. The purulent types occurred in children under 2 years of age in all but six instances (70 per cent.), and the tubercular cases in children under 2 years in only eleven instances (25 per cent.). One case, due to the staphylococcus, was an infant 12 days old. Two of the streptococcus cases followed fracture to the base of the skull, the other two followed chronic otitis media.

Meningismus may be seen in almost any acute febrile condition. In one case here described lumbar puncture gave issue to a cerebrospinal fluid under tension, but otherwise normal. In tubercular cases the tubercle bacillus can be found in the spinal fluid in nearly every case with the first fluid obtained.

A. NINIAN BRUCE.

A LARGE EXTRA-DURAL HÆMATOMA CONFINED TO THE
(35) **FRONTAL REGION.** (Volumineux hæmatome extra-durémérien exclusivement frontal.) ROCHER and LOGRE, *Rev. Neurol.*, 1916, Oct., pp. 253-257.

UNDER this heading is recorded a case which presented signs and symptoms of cerebral contusion with possibly some superadded meningitis, following a fall. There was no indication for surgical interference.

The post-mortem findings revealed a voluminous subdural hæmatoma. There was a fracture of the anterior fossa of the skull and a rupture of an important ramus of the anterior branch of the middle meningeal artery. No meningitis.

The author adds a plea for exploratory surgical measures in all cases of cranial trauma which do not appear to be doing well, even in the absence of localising symptoms. O. P. NAPIER PEARNS.

TWO UNUSUAL CASES OF CEREBRAL HÆMORRHAGE. OLIVER
(36) LATHAM, *Reports from Path. Lab. of Lunacy Depart., New South Wales Government*, 1916, iii., pp. 160-163.

THREE cases are actually described.

Case I.—A man, aged 31, was struck by a stone when 12 years of age and knocked down, but did not lose consciousness. Shortly afterwards his right arm became paralysed, and his right leg stiff. At 19 he took fits, at first mild, afterwards violent. Later he was certified insane because of his fits, refusing food, using obscene language, and crying out loudly. He died in an

epileptiform attack. Post mortem, a large and old hæmorrhage was found in the right internal capsule, external capsule, and adjoining nuclei up to the island of Reil. Part was calcified. The arterioles and venules round this area showed advanced hyaline degeneration. Numerous hæmorrhages were also found in the cerebellum.

Case II.—A man, aged 31, was admitted to hospital with a history of sudden development of fits. Previously he had always been well. He refused food, was violent, used vile language, and was faulty in habits. Case diagnosed as acute mania. He died suddenly. Post mortem, a recent hæmorrhagic softening was found at the extreme tip of each occipital lobe. The rest of the brain appeared normal. Advanced chronic nephritis was present. The Wassermann reaction was negative a few hours after death. The vessels surrounding this area showed proliferation of the lining endothelium, but not yet hyaline degeneration.

Case III.—A man, aged 60, who had been in hospital for twenty-five years with obscure delusions of persecution and determined idleness, suddenly had a succession of obscure seizures, became confused, and afterwards improved. His mental state then became quite reversed, he began to work hard, and accused himself of all he had formerly accused others. Post mortem, only a very mild degree of hyaline degeneration was seen in the brain and cord, and the cervical and lumbar, but not the dorsal, regions contained hundreds of capillary hæmorrhages.

A NINIAN BRUCE.

PARALYSIS OF THE UPPER FACIAL IN HEMIPLEGIA. (La (37) *paralysie du facial supérieur dans l'hémiplégie cérébrale.*) P. LE DAMANY, *Presse Médicale*, 1917, Jan. 4, No. 1, p. 1 (1 fig.).

AFTER a good account of the familiar theories on the relative escape of the upper facial movements in hemiplegia, the writer expresses his belief that their cortico-nuclear path is by way of the ansa lenticularis. He records a case in which necropsy proved that a unilateral lesion of the brain can produce a complete palsy of upper facial movements: the ansa was destroyed by a focus of hæmorrhage. He says that the fact of the separate cortical origin of the fibres for the upper facial movements, and their path by the anterior part of the lenticular nucleus and the ansa lenticularis, enables us to understand how a unilateral lesion can, according to its site, affect sometimes the lower facial movements alone, and sometimes all the facial movements. But we are still ignorant, he says, why the lower facial movements are always involved, and why an isolated palsy of the upper facial movements is never seen in paralysis of central origin.

LEONARD J. KIDD.

A REPORT OF AN UNUSUAL CASE OF CEREBRAL MALARIA.

(38) OTTO T. BROSIUS, *Journ. Amer. Med. Assoc.*, 1917, lxxviii., Jan. 13, p. 106.

A GIRL, aged 11 years, developed in Panama paralysis of the right side of the body, following two attacks of convulsions. Malaise, headache, and high fever had been present for the previous two days. Lumbar puncture gave issue to a clear fluid which contained nothing abnormal. A blood smear showed crescents and a very few ring forms. The white blood count was 12,800. The hemiplegia had disappeared on the second day in hospital, although the limbs affected remained weak.

According to Manson, cerebral malaria was at first supposed to have been caused by the malarial pigment which formed thrombi in the cerebral veins, but later investigation gave emboli formed by the parasites as the etiologic factor in this condition.

A. NINIAN BRUCE.

TUBERCLES OF THE PONS. (Tubercles de la protubérance.)

(39) D'ESPINE and DEMOLE, *Rev. Neurol.*, 1916, Aug.-Sept., pp. 176-179.

THE case described in this article showed, some months before death from tuberculous meningitis, facial and ocular paralyses on the right side, increased knee jerks, and double ankle clonus.

At the post mortem these special signs were found to be due to two discrete tubercles, one in the floor of the fourth ventricle involving the nucleus of the sixth nerve and the course of the seventh, and the other among the pyramidal fibres of the pons.

Tuberculous lesions were also found in the lungs, intestines, and cerebral organs.

O. P. NAPIER PEARN.

ON PARTICIPATION OF BOTH CEREBRAL HEMISPHERES IN

(40) **THE FUNCTION OF LANGUAGE.** (*Sulla compartecipazione di ambedue gli emisferi cerebrali alla funzione del linguaggio.*)

F. UGOLOTTI, *Riv. di Patol. nerv. e ment.*, 1916, xxi., p. 539.

A RIGHT-HANDED man had an ictus followed by verbal deafness and sensory aphasia at the age of 43. About three months later the symptoms became worse, and necessitated his being sent to an asylum, where he died four years later of pulmonary tuberculosis at the age of 47. During the first stage of his disease the verbal deafness and sensory aphasia were incomplete.

In the second stage his capacity for understanding rapidly

diminished and finally disappeared together with his faculty of speech.

Post mortem serial sections showed two fundamental lesions, almost identical, one in the left and one in the right hemisphere. In the left hemisphere was a softening affecting the cortical substance of the posterior two-thirds of the first temporal convolution, the posterior lobe of the insula, and in a very slight degree the operculum of the ascending parietal convolution.

In the right hemisphere was a softening of the posterior half of the first temporal and in a very slight degree of the second temporal and of the operculum of the ascending parietal. The softening of the left first temporal was the cause of the sensory aphasia, and supervention of softening in the right first temporal some months later was the cause of the total aphasia.

J. D. ROLLESTON.

A CASE BEARING ON THE FUNCTION OF THE PITUITARY

(41) **BODY.** WILLIAM BOYD, *Journ. Amer. Med. Assoc.*, 1917, lxviii, Jan. 13, p. 3 (2 figs.).

THE case of a boy, aged 10½ years, who began to suffer from slight attacks of frontal headaches and vomiting. Later temporal hemianopsia developed in the right eye; the left eye showed no field of vision. X-rays showed a slight enlargement of the sella turcica. The tolerance to sugar was increased, but there was no polyuria. Operation was attempted, and a small amount of a soft translucent growth was removed by Dr Cushing. The patient died about a month later with symptoms of increased intracranial pressure and cerebral irritation.

At the autopsy the pituitary body was found to be normal and lying free in the sella. In the interpeduncular space, and apparently growing from the region of the floor of the third ventricle, a soft greyish mass was found. The left optic nerve was embedded in the mass, which also completely surrounded the infundibular stalk, and constricted it as if it had been a ligature. It proved to be a glioma.

As the symptoms were those of a lesion of the posterior lobe of the pituitary, and the effect of the tumour had been to interfere with any communication which may normally exist between the posterior lobe and the third ventricle, the author regards the case as favouring the theory that the secretion of the posterior lobe of the pituitary passes by way of the infundibular stalk into the third ventricle.

A. NINIAN BRUCE.

- SYPHILITIC THROMBOSIS OF THE BASILAR ARTERY OF**
 (42) **THE BRAIN, WITH REMARKS ON SYPHILITIC LEPTOMENINGITIS, AND ON A SYPHILITIC CASE IN WHICH THE PITUITARY GLAND WAS AFFECTED.** J. PARKES WEBER, *Clin. Journ.*, 1916, xlv., p. 405.

Case I.—A man, aged 36, was admitted to hospital for headache, especially on the right side. Wassermann's reaction was positive. Six days after admission, he suddenly developed right hemiplegia and aphasia, and died in coma the next day.

The necropsy showed thrombosis of the anterior portion of the basilar artery, and a scar in the liver. Macroscopically, there was no meningitis visible, but microscopy showed syphilitic endarteritis obliterans of the basilar artery, and the two posterior cerebral arteries.

Case II.—The patient was a man, aged 44, who had contracted syphilis seventeen years previously, and who suffered from transient epileptiform attacks, one of which proved fatal.

For some months before death there was temporary polyuria.

Post mortem, in addition to some periarteritis of the pia mater at the base of the brain, there was a similar affection of the pars intermedia of the pituitary body.

The writer reviews the literature, including a recent case reported by him of diabetes insipidus in a boy with positive Wassermann's reaction (*c. Review*, 1912, x., p. 586).

J. D. ROLLESTON.

- A PROBABLE CASE OF NERVOUS HEREDO-SYPHILIS. (Hérédosyphilis nerveuse probable.)** L. BABONNEIX, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1516.

THE patient, a man aged 30, showed a great variety of nervous symptoms, the chief of which were of a tabetic and cerebellar character, while symptoms of disseminated sclerosis, chorea, paralysis agitans, and pyramidal lesions were also present.

J. D. ROLLESTON.

- AN EXAMINATION OF THE BLOOD SERUM OF IDIOTS AND IMBECILES BY THE WASSERMANN REACTION.** J. FROUDE FLASHMAN and OLIVER LATHAM, *Reports from Path. Lab. of Lunacy Department, New South Wales Government*, 1916, iii., pp. 73-78.

THE blood of every individual inmate of the Newcastle Hospital for the Insane, an institution which receives only idiots and imbeciles, was examined for the Wassermann reaction. This

comprised 436 cases. Of these, 29 gave an absolutely positive reaction, and 14 were doubtful, but probably syphilitic, making a total of 43, or 9·8 per cent.

525 subjects were also similarly examined in the idiot and imbecile population of the Hospitals for Insane at Newcastle, Rabbit Island, and Stockton. 11 per cent. full positive reactions were obtained, and 7 per cent. partial reactions.

About 32 of the 95 positive and part positive reactions showed evidence of syphilitic stigmata, *e.g.*, 11 per cent. showed Hutchinson's teeth and interstitial keratitis.

Previous observers had reported the following results:—

Rabiat and others examined 246 cases, and found that 76, or 30·8 per cent., were positive; Kellner and others, in 216 cases, found 16, or 7·4 per cent., positives; Liepmann examined 78 cases, with 7 positives, or 9 per cent.; Dean reported 51 positive out of 330 cases, or 15·4 per cent.; while the figures for Dalldorf Asylum were 13·2 per cent.

A. NINIAN BRUCE.

THE ANTAGONISTIC ACTION OF NEGATIVE SERA UPON THE

(45) **WASSERMANN REACTION.** A. W. SELLARDS and G. R. MINOT, *Journ. Med. Research*, 1916, xxxiv., May, pp. 131-147.

SERA which give a negative reaction with the Wassermann test possess definite inhibitory properties towards positive syphilitic sera, except in certain special cases.

The extent of this inhibitory action in negative sera varies widely in different diseases, but it is usually comparatively weak. It is easily demonstrable, even though it is present only in slight degrees.

Human sera present three distinct phases in their behaviour toward the complement fixation of the Wassermann reaction, *i.e.*, a (1) negative, (2) positive, and (3) inert action.

These results are explained most readily on the basis of a balanced mechanism. The inhibitory action of negative sera cannot be accounted for solely on the basis of its content in natural sleep amboceptor.

A. NINIAN BRUCE.

SURFACE-TENSION AND THE WASSERMANN REACTION.

(46) VINCENT B. NESFIELD, *Lancet*, 1917, i., Jan. 6, p. 18.

THE author considers (1) that surface-tension is the all-important factor in the Wassermann reaction; (2) that alcohol, bile, carbolic acid, and solutions of cholesterin, which act as antigens in the Wassermann test, all have very low surface-tension and greatly reduce the surface-tension of serum; and (3) it is difficult to distinguish a syphilitic antigen from a substance which lowers surface-tension.

A. NINIAN BRUCE.

THE REACTION OF THE CEREBRO-SPINAL FLUID IN THE
(47) **PSYCHOSES.** PAUL G. WESTON, *Journ. Med. Research*, 1917,
xxxv., Jan., p. 367.

THE total number of fluids examined was 105. 73 were from cases of paresis, 8 from cases of organic dementia, 7 from cases of dementia præcox, 10 from cases of manic-depressive psychoses, and 7 from cases of imbecility.

The results are tabulated and are in accord with the statement of Mott that the reaction of the cerebro-spinal fluid varies only slightly in different conditions. The method used was that of Bisgaard who determined the hydrogen ion concentration of the fluid, and gave the pH value as 8.10. The greatest degree of alkalinity here found was pH=8.3, and the lowest pH=7.9. The variation was practically the same in each group. The average for all cases was pH=8.12.

A. NINIAN BRUCE.

THE RETINITIS OF ARTERIO-SCLEROSIS, AND ITS RELATION
(48) **TO RENAL RETINITIS AND TO CEREBRAL VASCULAR**
DISEASE. R. FOSTER MOORE, *Quart. Journ. Med.*, 1916-17, x.,
p. 29.

THE changes in the retina and its vessels which are indicative of arterio-sclerosis are defined, and their development and course followed over considerable periods of time.

It is concluded that there is a form of retinitis which is associated with severe general arterio-sclerosis; it is caused by the local retinal vascular disease, and its association with disease of the kidney is only incidental. It is in large measure distinct from renal retinitis in its ophthalmoscopic characters, in its significance, and in its prognostic value.

It is frequently unilateral, and cotton-wool patches never occur. Its gradual evolution from a condition of retinal arterio-sclerosis can be traced. The tenure of life of subjects affected is uncertain, but they may live a number of years, and death is referable to disease of the vascular system, and not of the kidney.

The relation between disease of the cerebral and of the retinal arteries is striking and conclusive. Of 44 patients suffering from a gross cerebral vascular lesion, 31 (70 per cent.) exhibited evidence of retinal vascular disease, and in 19 of them (43 per cent.) it was severe in degree. Of patients in whom retinal vascular disease was sufficiently severe to give rise to symptoms, in 46 gross cerebral lesions developed. Of these 46, 21 (46 per cent.) had either suffered from such a lesion or developed one in about three years; 18 of the remainder are still alive.

A. NINIAN BRUCE.

**THROMBO-ANGIITIS OBLITERANS (NON-SYPHILITIC ARTER-
(49) ITIS OBLITERANS OF HEBREWS) AFFECTING THREE
LIMBS.** F. PARKES WEBER, *Proc. Roy. Soc. Med.*, 1916, x., Dec.
(Clin. Sect.), pp. 1-3. (Discussion.)

THIS case is one of those referred to in the author's previous paper upon this subject (*v. Review*, 1916, xiv., Dec., p. 508). In 1908, when the patient was 31, his right leg was amputated below the knee for commencing gangrene. The disease had commenced two years previously with pains of the "intermittent claudication" type. The posterior tibial artery showed organised thrombus, the upper part showed no thrombus, but narrowing of the lumen from a kind of endarteritis obliterans; one of the *venæ comites* of the middle part of the anterior tibial artery was also obliterated by organised thrombus.

In July 1912, owing to great and unyielding pain in the right stump, a second amputation through the middle of the femur had to be performed.

In September 1916 he was again admitted to hospital with "intermittent claudication" of the left leg. There was no pulsation in the dorsalis pedis artery nor in the left radial artery.

A. NINIAN BRUCE.

**A STUDY OF THE METABOLISM IN A CASE OF AMYOTONIA
(50) CONGENITA.** F. POWIS and H. S. RAPER, *Quart. Journ. Med.*,
1916-17, x., pp. 7-19.

THIS case was that of a girl, aged 4 years, who was under observation from 2nd June till 15th November 1915.

The chief findings were:—

A diminution of hepatic functional activity as manifested by the presence of acholia. Whether this is an accidental association or not is uncertain; a normal calcium retention, associated with a relatively high potassium retention; a low creatinine excretion as established by previous observers, and accompanying this a relatively high creatine excretion (*v. Review*, 1916, xiv., p. 257). Until more is known about the formation of creatine, its rôle in the body, and the reasons for its appearance normally in the urine of children, this high creatine excretion cannot be explained. Treatment with bile salts or dried ox bile produced some improvement, as manifested by an increase in strength of the muscles, and a change towards the normal in the ratio of the potassium to the calcium retention, the normal values, owing to a lack of other data, being deduced from the ash analyses of young animals.

A. NINIAN BRUCE.

BERI-BERI. G. MARSHALL FINDLAY, *Practitioner*, 1917, xcvi., Jan., (51) p. 69.

BERI-BERI is accurately described in Chinese documents of the second century, and it is probable it is referred to in writings as early as 2697. The word itself is derived from a Singhalese word meaning "weakness." Strabo, writing in the third century A.D., describes an epidemic as having occurred among a Roman army in Arabia in B.C. 24. Modern interest dates from the seventh century, when Dutch physicians came in contact with the condition in the East Indies. There appear to be three markedly endemic centres, one embracing Japan and Eastern China, the second including the Dutch East Indies (Java, Sumatra, and Borneo), the Malay Peninsula, and the Philippine Islands, and a third which involves Brazil. The disease is also prevalent, though to a less extent, in India, Northern Australia, and on the coast of Africa. It is most common in sea-girt islands, and on the shores of continents; it rarely penetrates into the hinterlands.

The present article is based on a series of thirty-six cases which have lately occurred in certain ships on the Egypt and East Indies station. At one time the health of the Japanese Navy was seriously impaired by the ravages of this disease. The men's rations, at this time, consisted almost entirely of white or polished rice, but by decreasing the rice, and increasing the other proteid constituents of the diet from 109 to 196 gm., the disease was almost entirely eliminated. The author's opinion is that beri-beri is produced whenever a diet free from vitamines is persisted in for any length of time, and is thus analogous to myxœdema.

A. NINIAN BRUCE.

CALCIUM AND EPILEPSY. GUY P. U. PRIOR and S. EVAN JONES, (52) *Reports from Path. Lab. of Lunacy Department, New South Wales Government*, 1916, iii., pp. 122-133.

A DIMINISHED amount of free calcium is found in the blood of epileptics, also a diminished excretion. Epileptics may be benefited by giving them calcium, especially if combined with bromides. Menstruation stimulates fits by promoting calcium change, and the number of fits may be diminished by increasing the calcium index by hypodermic injections. During the period under review, the total number of fits in the patients was less than the average for a similar period by 524, a reduction of 31 per cent. The number of cases here recorded is eight women and four men.

A. NINIAN BRUCE.

TWO CASES OF PARKINSONIAN HEMIPLEGIA. (Hemiplegia
(53) *Parkinsonia. Duas observações.*) E. VAMPERE, *Ann. Paul de
med. e cir.*, 1916, vi, p. 126.

THE patients were aged 40 and 45 respectively, in whom the
movements were exclusively right-sided. J. D. ROLLESTON.

CASE OF PULMONARY HYPERTROPHIC OSTEO-ARTHROPATHY
(54) **OCCURRING IN A CASE OF CONGENITAL HEART DISEASE.**
H. BATTY SHAW and STANLEY MELVILLE, *Proc. Roy. Soc. Med.*,
1916, x., Dec. (Clin. Sect.), pp. 8-11.

A CASE of this rare condition in a man, aged 32. Apart from
well-marked emphysema and slight bronchitis, the lungs seemed
normal. A systolic murmur was heard over the heart, conducted
in every direction, and thought to be due to a patent inter-
ventricular septum associated with occlusion of the pulmonary
artery. The Wassermann reaction was negative.

A. NINIAN BRUCE.

PSYCHIATRY.

RECORDS OF SPEECH IN GENERAL PARALYSIS. E. W.
(55) SCRIPTURE, *Quart. Journ. Med.*, 1916-17, x., pp. 20-29 (9 figs. and
1 plate).

ABOUT twenty cases were studied, ranging from those with no
speech defect which could be detected by the ear to those far
advanced towards dementia. Only the milder cases are described
here. The apparatus consisted of a revolving cylinder covered
with smoked paper and a recording tambour. A complete analysis
of a single sentence can now be made in about five hours, or with
an assistant in two hours. Records are shown of the words
"hippopotamus" and "Peter Piper's peppers," first in the normal,
and second in the paretic. Since speech is the most delicate,
sensitive, and complete method of expressing mental activity, it
is natural to find in the speech of paretics the best method of
studying the mental disturbance in this disease. The speech
records show traces of practically the entire mental derangement
of the paretic, and will detect lack of precision in the speech
where it is quite impossible to detect any abnormality by the
ear. For this lack of precision the term "*asaphia*" is suggested
as better than "cortical ataxia." *Asaphia* never appears in
neurasthenia, psychasthenia, mania, or melancholia. Its presence
in a speech record is thus strongly suggestive of general paralysis.
The limitation from arterio-sclerosis and diffuse syphilis of the
brain is not yet clear.

Asaphia produces unprecise movements, ataxia produces irregular movements, and apraxia produces wrong movements, or movements which do not correspond to the intended purpose, although they may be correct for another purpose.

A. NINIAN BRUCE.

SHALL WE TREAT THE PARETIC? C. EUGENE RIGGS and E. M. (56) HAMMES, *Journ. Amer. Med. Assoc.*, 1917, lxxviii., Jan. 20, pp. 194-198.

BOTH clinically and biologically it has been demonstrated that treatment is a material benefit to the paretic. Before the salvarsan era, the number of remissions varied from 4 to 20 per cent. Since its use, and the employment of modern forms of therapy, they have been greatly increased in frequency. Treatment, to be effective in paresis, must be effective in its earlier stage; in cases of long standing it is useless; cell destruction cannot be replaced. Also, to be effective, it must be persistent. It may be a matter of many months before the activity of the infection can be influenced.

A. NINIAN BRUCE.

THE CLINICAL AND ANATOMICAL FEATURES IN ALZHEIMER'S DISEASE AND RELATED CONDITIONS. C. I. (57) LAMBERT, *Psychiat. Bull. of New York State Hospitals*, 1916, ix., Oct., p. 413.

IN 1906 Alzheimer described an atypical form of mental disorder occurring in middle life, insidious in its development, rapid in its course, and reaching a profound degree of dementia. Aphasia, asymbolic, and apraxic disturbances occur, which are all the more striking in the absence of definite paralytic phenomena.

Pathologically a widespread peculiar degeneration of nerve cells occurs, showing in altered stainability, thickening, and coalescence of the neurofibrillæ, and in knarled, skeletal neurofibrillæ rests, the plasmatic substance having dissolved and disappeared from the nerve cells. In addition numerous plaques may be seen scattered throughout the cortex.

In this paper Lambert gives clinical summaries of five interesting cases of the type described above, and discusses in considerable detail the pathological findings. It is principally the cells of the second and third layer of the cortex which are affected, the large pyramidal cells being seldom involved. The fibril alteration seems to take place initially in the individual fibrils, and usually in those portions of the cell where pigment is deposited. The change is presumed to be of a chemical nature as the fibrils easily take silver stains. The similarity of these changes to those occurring in senility and senile dementia is remarked on, but quantitatively both the gross and microscopic changes are much more marked

than in the senile cases. The atrophy appears to develop irrespective of vascular distribution, and this leads one to suppose that some abiotrophic process underlies the condition. The difficulties of clinically differentiating Alzheimer's disease from cases of senile dementia, arterio-sclerotic brain disease, and general paralysis is emphasised. A review is given of the work of others, and an excellent series of plates showing the pathological findings is appended.

D. K. HENDERSON.

**SOME CLINICAL DEDUCTIONS BASED ON THE OCCURRENCE
(58) OF ACETONE IN THE URINE OF THE ACUTELY INSANE.**

G. H. MILLS and R. G. HEARNE, *Psychiat. Bull., New York State Hos.*, 1916, ix., Oct., p. 433.

IN a number of acutely insane patients who died suddenly the authors found prior to death casts and albumin in the urine, and post mortem various kidney changes which in some cases seemed to be the most important finding. In addition it was found that acetone was not infrequently present before the casts and albumin appeared. It was consequently thought that some kidney toxin or irritant was at work, and the presence of acetone was used as an indicator for treatment. Treated as nephritics these cases gave unsuccessful results, but from a gastro-intestinal auto-intoxication standpoint fine success was attained. Clinical records are given of nine fatal cases, all of whom showed certain symptoms in common, viz., physical reduction and gastro-intestinal symptoms; the mouth was foul, the tongue coated, the bowels constipated, fever was present, and restlessness and sleeplessness.

Twenty-seven other similar cases have now been studied, of whom twenty-three recovered and four died.

The treatment consists of free catharsis with calomel, salts, &c., free washing out of the lower bowel by enemas, saline irrigations, or solutions of sodium bicarbonate; forced feeding if necessary; the use of intestinal antiseptics, preferably either thymol or beta-naphthol. Eggs and broth are usually forbidden.

D. K. HENDERSON.

**THE MENTALITY OF ARISTOCRACIES AND THE STUDIES OF
(59) THE ALIENIST, PAUL JACOBY. (La mentalità delle aristocrazie e gli studi dell'alienista Paolo Jacoby.)**

FR. DEL GRECO, *Riv. ital. de Neurop., Psichiatr. ed Elettrotet.*, 1916, ix., p. 391.

A REVIEW of the Russian alienist's work, "Etudes sur la selection chez l'homme," the second edition of which was published in 1904. In the first part of the book which is entitled "Power" Jacoby shows that human aristocracies soon degenerate in their physical organism from father to son and become extinct. The cause

is to be found in the excess of nervous labour expended in exercising a high social function. Jacoby reconstructs the medico-psychological history of the Cæsars and their families and then that of many other royal houses. He demonstrates the rapid extinction of their descendants in association with insanity and physical and moral abnormalities.

In the second part of the book entitled "Talent" Jacoby enunciates a more general idea. He shows that with the increase of civil life there is an emigration of the population from the country to the towns. It is in these foci of intense and devouring toil that distinguished men arise who from the beginning have a certain nervous and mental lack of balance. They reach their culminating point in valuable social work, whether theoretical or practical, but their physical descendants become extinct, lead evil lives, or degenerate.

J. D. ROLLESTON.

MENTAL REGRESSION: ITS CONCEPTION AND TYPES. F. L. (60) WELLS, *Psychiat. Bull., N.Y. State Hos.*, 1916, ix., Oct., p. 445.

REGRESSION takes place when fundamental trends are replaced by trends which are less fundamental; a typical example of this is seen when the conventional young woman, disappointed in love, betakes herself to a convent, in this case love being replaced by religion.

The sexual-parental, the trends for obtaining food, those for protection against enemies, and the social trends, *i.e.*, those for co-operation with one's kind, are considered as fundamental. One fundamental trend superseding another is not regression. Each person must have a certain minimum in meeting fundamental trends, and when a less important trend absorbs energy from a fundamental one regression takes place. As a person matures his trends change so that what was normal for a child is abnormal for the adult, *e.g.*, day-dreaming, which is essentially a reversion towards the infantile. The author then contrasts this conception of regression as a reversion to the infantile with the *quietistic* conception of Ribot, which lays emphasis on regression as representing an economy of energy, *e.g.*, "shirking" reactions.

Auto-erotic, or what the author prefers to call *auto-hedonic* habits, *e.g.*, nail-biting, thumb-sucking, anal-eroticism, and masturbation, are first discussed, and it is shown how in the above types of regression *organic* satisfaction is almost immediately attained.

In other types, however, organic satisfaction is less immediate and may be impossible to trace. When regression is thought of as a return to the infantile this means that it is a return to

protection and domination, and whatever persons or things play these rôles are symbols for father and mother.

The following main groups are then discussed in detail:—(1) A regression which emphasises dependence upon, and domination by, some outside influence (naturally symbolised by the “father”—relationship); (2) a similar regression in which the dominant force is figured as “maternal”; (3) the overcoming of the regressive tendencies, figured as a *resurrection* or *re-birth*; (4) a regression to protection, not only by a mother-representative, but also by a deified culture-hero (founders of religions, Christ); (5) a regression consisting, not in a reversion to the infantile, but in the symbolisation of adult trends (usually erotic: “bride of Christ”); (6) the limit of introversion, symbolised by a return to the mother-representative as in (3), but without “re-birth” therefrom.

The whole article is written clearly and convincingly, and well repays careful reading. Regression, like fever, is a defence mechanism; but again, like fever, it uses up energy that in health would go elsewhere.

D. K. HENDERSON.

SOME NOTES ON TEST MEALS IN THE INSANE. OLIVER

(61) LATHAM, *Reports from Path. Lab. of Lunacy Department, New South Wales Government*, 1916, iii., pp. 105-119.

TWENTY-FOUR cases of insanity were studied. In eleven test meals from five normal people, free HCl averaged 27.5 and combined HCl 6. In the insane cases the average free HCl was 9.3, and the combined HCl 12.6. Not only was the total HCl thus reduced, but the ratio of free to combined, which is greater than four to one in normal cases, becomes less than one to one in these special cases of insanity.

A. NINIAN BRUCE.

TREATMENT.

THE ACTION OF DIGITALIS IN PNEUMONIA. ALFRED E. COHN

(62) and ROSS A. JAMIESON, *Journ. Exp. Med.*, 1917, xxv., p. 65.

DIGITALIS acts during the febrile period of pneumonia. It produces a beneficial, possibly a life-saving effect in cases of auricular irregularity (fibrillation and flutter). Whatever beneficial action it has on the function of the normally beating non-febrile heart may be expected from its use in the febrile heart in pneumonia.

A. NINIAN BRUCE.

Reviews

TEXT-BOOK ON NERVOUS DISEASES. Edited by Dr HANS (G3) CURSCHMANN, Physician to the St Rochus Hospital in Mainz. Translated under the direction of CHARLES W. BURR, M.D., Professor of Mental Diseases, University of Pennsylvania. 246 figures. Two octavo volumes, cloth, \$12.00. One octavo volume, cloth, \$8.00. P. Blakiston's Son & Co., Philadelphia.

THE days when it is possible for a text-book covering the whole field of nervous disease to be produced by one person appear to be drawing to a close on account of the vastness of the field requiring to be covered, and the rapidity with which our knowledge of this particular branch of medicine is increasing. Probably Professor Oppenheim's well-known text-book represents the best example of such a text-book; it covers the whole field of neurology, it analyses and criticises with remarkable clearness old and recent views, it incorporates all recent advances, it is abundantly supplied with references to literature, and contains the unrivalled personal, clinical, and pathological experience of one who has devoted his life to the elucidation of nervous disease.

In place of the individual text-book, however, there is gradually coming into vogue the text-book in which each chapter is handed over to someone who has specialized in that particular disease. Such a book possesses the advantages of highly specialized opinions, but suffers usually from a certain amount of unavoidable repetition and variability in the different chapters.

The present text-book is an example of this latter type, and aims at producing for students and practitioners a book which contains all that is now accepted as established, as well as more recent advances in treatment, although devoid of that fulness to be found in special monographs. It is divided into two volumes. The first deals with general diagnosis, diseases of the peripheral nerves, spinal cord and brain, and the myopathies. General diagnosis is treated by Schoenborn, the peripheral nerves by Steinert, the normal and pathological physiology of the spinal cord by Rothmann, disseminated sclerosis and syringomyelia by Schlessinger, focal diseases of the cord by Finkelnberg, the myopathies by Curschmann, and diseases of the brain by Liepmann. In the second volume, diseases of the meninges are treated by Starck, cerebral tumours, abscesses, and non-purulent encephalitis by Lewandowsky, disturbances of the circulation of the brain by Hirsch, general paralysis and epilepsy by Gaupp, organic diseases of childhood by Ibrahim, hyperkinetic diseases by Pineles, psychasthenic states by Aschaffenburg, diseases of the

sympathetic system by Müller, vasomotor and trophic diseases by Curschmann, intoxication diseases by Quensel, and operative conditions by Krause, while the final chapter on neurasthenia, psychasthenia, and borderland mental states is the work of Dr C. W. Burr, the translator.

The value of this translation is that it presents in English a good account of the present German views on nervous disease as described by the investigators themselves. The disease pictures are good, and italics are frequently used to accentuate more important headings and definitions. The opinions expressed and the illustrations used are almost all of German origin, and being the personal and matured experience of men of established reputation, the result is an excellent description of modern German thought.

The translation has been on the whole well done. There is a good index, evidently prepared with much care, and the illustrations are well reproduced. No references to literature are given.

RESEARCHES ON RHEUMATISM. F. J. POYNTON, M.D., and (64) ALEXANDER PAINE, M.D. Pp. xi+461, with frontispiece in colour and 106 illustrations. J. & A. Churchill, London. Pr. 15s. net.

THE names of Poynton and Paine are definitely established in the history of rheumatism. Their contributions extend over a period of fifteen years, and are scattered through many different medical journals. In this volume, however, they have collected together their principal papers, and arranged them more or less in the order of their publication. The experimental studies are given in detail, and the papers are linked together by prefaces, which indicate the line of thought and the new facts that each contains. The book concludes with a special article describing the bearing of these investigations upon clinical medicine and public health.

The various contributions are grouped under three headings. The first contains seven papers, all written before the isolation of the *Diplococcus rheumaticus*. The second contains those papers written after the isolation of the *Diplococcus rheumaticus*, and which demonstrate the etiology and inquire into the nature of the rheumatic lesions, both human and experimental. It includes an important paper which was published in 1906, in which a number of cases of fatal chorea were investigated, and the organism discovered in the pia mater and in the adventitial sheath of vessels in the cerebral cortex. The third part is new matter. It consists of a summary of our present knowledge of acute rheumatism based on the previously described work, and discusses in turn

etiology, pathology, symptomatology, diagnosis, prognosis, treatment, and prevention.

The organism to which the authors attribute the disease is the *Diplococcus rheumaticus*. It is a small micrococcus about $0.5\ \mu$ in diameter, and grows usually in pairs or in short chains. It does not retain Gram's stain with great tenacity. It will live for long periods of time on blood-agar, and retains its vitality in the dry state for many months. Its saprophytic stage is characterised by rapid loss of virulence.

The investigation of rheumatism presents many difficulties. Although the acute disease in childhood is rife in this country, fatal cases are not very frequent, and thus progress is necessarily slow. The micrococcus is also rapidly destroyed by the living tissues, and the favoured sites, namely, the pericardium, synovial membranes, and brain, are closed cavities, and not in communication with the exterior as in tuberculosis. The infection is also moulded on the pyæmic type, and produces essentially local lesions, which, combined with the great resistance of the tissues, makes it difficult to isolate the micrococcus from the exudations even if their presence be obvious from films. Living in the local lesions, their isolation from the blood is not to be, as a rule, expected, and their demonstration in the tissues is not easy, especially as they are so minute, and stain so badly. "We would point out that we have never relied upon the production of arthritis in rabbits as a proof of rheumatism." The organism may be isolated from the diseased tonsils of cases of rheumatism, and has been found to be identical with that isolated from acute rheumatic lesions.

The authors insist upon the fact that the acute rheumatism of childhood is a definite clinical entity. "Among the many criticisms that have been made upon the investigations, the definite objection that acute rheumatism is not a specific disease we have been prepared to meet with determination and conviction by the statement that it is one of the most special and definite diseases in this country." The establishment of the cause of a most important disease amongst those classed as "rheumatic" is the first step in the study of the problem, and clears up much disputed ground. Three observations in the problem of arthritis are of particular importance: one is, that the same infective agent may produce all varieties of non-suppurative arthritis, from a simple synovitis to an osteo-arthritis; another, that the acute and very chronic lesions produced by experiments may be associated with effusions that are sterile; and the third, that an intravenous infection may produce a monarticular arthritis. Although the organism possesses a proclivity for attacking certain tissues, there is no theoretical barrier to its attacking any tissue, and broncho-pneumonia, nephritis, peritonitis, or appendicitis may all be so produced.

Probably all grades of virulence also exist, and the disease may show itself clinically in all forms, from the most acute to the most chronic forms.

For further information the reader must be referred to the book itself, which he will find full of information. Whether these results will stand the test of time, or will require to be modified, it is not possible to say to-day; but it is possible to state that this work is pioneer work in a most difficult subject, and that this collection of papers does form a definite landmark in the history of rheumatism.

THE CATARRHAL AND SUPPURATIVE DISEASES OF THE
 (65) **ACCESSORY SINUSES OF THE NOSE.** ROSS HALL SKILLERN,
 M.D. Second edition, thoroughly revised. Pp. xxii + 417, with 287
 illustrations, some coloured. J. B. Lippincott Company, Philadelphia
 and London. Pr. 21s. net.

THE close connection between the accessory sinuses of the nose and the brain, and the liability of infective processes originating in the one to spread inwards to the other, make this book of importance to the neurologist. The first edition was published in 1913, and the fact that a second edition has so soon become necessary shows that it fulfils a definite need. This, the second edition, has undergone a systematic revision, and much new matter has been incorporated, including the treatment of sinus disease in children, the use of the naso-pharyngoscope in the diagnosis of obscure conditions in the posterior ethmoid and sphenoid regions; the diagnostic needle puncture of the maxillary sinus is more fully explained, and the possible dangers and means of avoiding them are pointed out; Canfield's operation on the maxillary sinus is compared with the preturbinal method, with instructions for and illustrations of both the immediate and ultimate effects of operations on the sinuses; a compilation of the American mortalities following the Killian operation on the frontal sinuses is given; a complete revision has been undertaken of the chapter on the sphenoid sinus, with description and illustrations of Halle's new operation; and a chapter on combined empyema or multiple sinusitis has been added. The book is divided into six parts, dealing in turn with general considerations, the maxillary sinus, the frontal sinus, the ethmoid labyrinth, the sphenoid sinus, and combined empyema. All are discussed very thoroughly, the illustrations being very numerous and well reproduced, and special attention is directed to the important subject of treatment, particularly to that phase which deals with the judgment of the attending surgeon in regard to the procedures which should be followed under different circumstances, *e.g.*, the proper moment to operate, and what form of operation is indicated, while the after-treatment following opera-

tion, a subject usually neglected in text-books, is also discussed together with the measures necessary to meet any untoward complication that may arise.

The object of the author to set forth in the English language a thorough and exclusive treatment of this subject has been fully attained here, and the book may thus be confidently recommended. Its value is enhanced by the addition of a large number of the more important references to the literature on this subject. There is a good index.

FORM AND FUNCTION. A contribution to the history of animal (66) morphology. E. S. RUSSELL, M.A., B.Sc., F.Z.S. Pp. ix+383, with 15 illustrations. John Murray, London. 1916. Pr. 10s. 6d. net.

THIS book is a brilliant piece of work although it contains no new matter. It is an historical account of the different attitudes which have been taken up from the time of Aristotle till the present day to the problem of the relation of function to form. Is function the mechanical result of form, or is form merely the manifestation of function or activity? What is the essence of life—organization or activity?

The main currents of morphological thought are three in number—the functional or synthetic, the formal or transcendental, and the materialistic or disintegrative. The first is associated with the great names of Aristotle, Cuvier, von Baer, and leads to the more open vitalism of Lamarck and Samuel Butler. The typical representative of the second attitude is E. Geoffroy St Hilaire, and this habit of thought has greatly influenced the development of evolutionary morphology. The third is not distinctly biological, but is common to many fields of thought. It dates back to the Greek atomists, and the success of mechanical science in the nineteenth century has induced many to accept materialism as the only possible scientific method. In biology it is more akin to the formal than to the functional attitude.

Owing to the great advances which modern biology has made in certain directions, its historical continuity has tended to disappear, and this book is an effort to trace the essential continuity of thought from the time of Aristotle to the present day, to show the gradual steps in the increase of our knowledge, to summarise more or less completely the contribution of each new worker, to define its value, and explain exactly in what way he contributed new facts to our understanding, and to show how the interplay of the different theories and the various swings of the pendulum in the gradual upward progress of our knowledge affected our views. It is here possible to discover the great names and landmarks in our knowledge, and to realise the significance of the contributions of Aristotle, Cuvier, Goethe, St Hilaire, Owen,

von Baer, Lamarck, Darwin, Haeckel, Gegenbaur, Samuel Butler, and very many others. The numerous different points of view are all clearly explained and interpreted.

The author does not express his own views on this subject, although he does not hide his strong sympathy with the functional attitude. He thinks that more insight will be gained into the nature of life and organization by concentrating on the active response of the animal as shown by its behaviour and in morphogenesis, than by merely giving exclusive attention to its structure, and by the realisation that living things are active, purposeful agents, not merely aggregations of protein and other substances.

It is to be hoped that the author will see his way later to supplement this work by giving us another describing his own attitude and views on this question.

HISTOIRE DES TRAVAUX DE LA SOCIÉTÉ MÉDICO-PSYCHO-LOGIQUE ET ELOGES DE SES MEMBRES. Dr ANT. RITTI, Secrétaire général de la Société. In two volumes. Masson et Cie, Paris. 1913-1914.

DR RITTI is to be congratulated upon the publication of these two volumes, in which there will be found an excellent history of the Medico-Psychological Society of France, together with a number of eulogistic tributes to the memory of many of its most distinguished members. The occasion for the publication of these was the occurrence of the fiftieth anniversary of the foundation of the Society, which was celebrated in June 1902.

The original conception of the Society appears to have come from Baillarger, physician to the Salpêtrière, and founder of the *Annales Médico-Psychologiques*. Renaudin, the superintendent of the Asylum at Fains, wrote to him in 1846 suggesting that he should give more attention to statistical researches in this journal. Baillarger replied that what was required was the establishment of a society of medical alienists, an idea which Renaudin strongly approved of, and was supported later by Aubanel, the superintendent of Marseilles Asylum. The result was that on 18th December 1847 a society was constituted in Paris, which in 1852 became the present French Medico-Psychological Society. The first meeting was held on 26th April 1852, when the members of committee were elected and details of administration arranged, and at the second meeting in June of the same year the first paper was read by Delasiauve upon mental confusion following epilepsy, and a discussion of this paper by Baillarger followed. The further proceedings of this Society are all faithfully recorded here, and form most interesting reading, especially in the light of our present knowledge.

After this there follows a large number of eulogies on distinguished members of the Society delivered at the annual public meeting, all of which conclude with a list of their principal publications. In an appendix to the first volume an account is given of the inauguration of the statue to Philippe Pinel in the *Place de la Salpêtrière* on 13th July 1885.

The second volume contains further eulogies, including one upon Baillarger himself, and allows us to realise how much work he accomplished during his life. The rest of the book is occupied by short biographical references of well-known members of the Society. In an appendix a description is given of the inauguration of the busts of Baillarger and of Fabret at the Hospital of the Salpêtrière in 1894, and of the inauguration of the busts of Pinel and d'Esquirol in the *Salle des Illustres du Capitole de Toulouse* in 1897. The volume concludes with an obituary of Professor Korsakoff of Moscow, delivered to the psychiatric section of the thirteenth International Congress of Medicine in Paris during August 1900.

It is no exaggeration to state that in the history of this Society, and in the activity of its members, we have recorded the history of psychiatry in France during the second half of the nineteenth century.

**REPORTS FROM THE PATHOLOGICAL LABORATORY OF THE
(38) LUNACY DEPARTMENT, NEW SOUTH WALES GOVERN-
MENT.** Vol. III. Pp. 203, with numerous illustrations. Wm. H.
Gullick, Sydney. 1916. Pr. 7s. 6d.

THE third volume of these reports contains fifteen papers, mostly the work of Dr Froude Flashman and Dr Oliver Latham, the director and the pathologist respectively of the pathological laboratory of the Lunacy Department of New South Wales. Most of these papers will be found abstracted in this number of the *Review*, and many are contributions of value. Dr A. W. Campbell contributes a paper upon the right cerebral hemisphere of a baby gorilla, which is a continuation of his well-known histological studies on the localisation of cerebral function. An interesting paper by Drs Flashman and Latham describes an unusual case of heterotopia of the spinal cord, discovered accidentally after death in a tabetic, and in another paper they record the results of the examination of the blood for the Wassermann reaction in a large number of mental defectives and imbeciles. Dr Latham records a case with malignant cervical glands, in which the spinal cord showed developmental abnormalities with primary degeneration of neurones, and another case of myasthenia gravis with hyperplasia of the interstitial tissues of the nervous system.

Review
of
Neurology and Psychiatry

Original Articles

**THE HISTOLOGY OF DISSEMINATED
SCLEROSIS.**

By JAMES W. DAWSON, M.D.

WITH A

PRELIMINARY COMMUNICATION

By the late ALEXANDER BRUCE, M.D., LL.D., and JAMES W.
DAWSON, M.D.

(With 48 Plates.)

IV.

HISTOLOGICAL STUDY.

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| <p>(1) INTRODUCTION: Clinical History of Case I.</p> <p>(2) STRUCTURE OF DIFFERENT TYPES OF AREAS:</p> <p> 1. An Actual Sclerotic Area:</p> <p> (a) Spinal Cord—</p> <p> (i) Nerve Fibres cut longitudinally.</p> <p> (ii) Nerve Fibres cut transversely.</p> <p> (b) Brain—</p> <p> (i) Nerve Fibres cut longitudinally.</p> <p> (ii) Nerve Fibres cut in various directions.</p> <p> 2. An "Early" Area:</p> <p> (a) Spinal Cord—</p> <p> (i) Nerve Fibres cut transversely.</p> <p> (ii) Nerve Fibres cut longitudinally.</p> | <p> (b) Brain—</p> <p> (i) Nerve Fibres cut longitudinally.</p> <p> (ii) Nerve Fibres cut in various directions.</p> <p> 3. Evolution of an Actual Sclerotic Area:</p> <p> A. Through Stages of increasing Glia Hyperplasia.</p> <p> B. Through a Stage of Fat Granule Cell Formation:</p> <p> (a) Spinal Cord—</p> <p> (i) Nerve Fibres cut transversely.</p> <p> (ii) Nerve Fibres cut longitudinally.</p> <p> (b) Brain—</p> <p> (i) Nerve Fibres cut in various directions.</p> <p> (ii) Nerve Fibres cut longitudinally.</p> |
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HISTOLOGICAL STUDY—*continued.*

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| <p>(2) STRUCTURE OF DIFFERENT TYPES OF AREAS—<i>contd.</i></p> <p>4. Other Types of Areas :</p> <p style="padding-left: 20px;">(a) Areolar Areas.</p> <p style="padding-left: 20px;">(b) Peri-vascular Sieve-like Areas.</p> <p style="padding-left: 20px;">(c) "Markschattenherde."</p> <p>(3) CHARACTERISTICS IN SPECIAL SITUATIONS :</p> <p>1. White Matter.</p> <p>2. Grey Matter.</p> <p style="padding-left: 20px;">(a) Central.</p> <p style="padding-left: 20px;">(b) Cortical.</p> <p>3. Peri-ventricular Sclerosis.</p> <p>4. Optic Nerve: Cranial and Spinal Nerve Roots, &c.</p> | <p>(4) CHANGES IN THE INDIVIDUAL TISSUE ELEMENTS :</p> <p>1. Nerve Fibres—</p> <p style="padding-left: 20px;">(a) Medullated Sheath.</p> <p style="padding-left: 20px;">(b) Axis Cylinder.</p> <p>2. Ganglion Cells.</p> <p>3. Neuroglia.</p> <p>4. Blood-vessels and Lymphatics; Cell Elements in the Adventitia.</p> <p>(5) OTHER HISTOLOGICAL FEATURES :</p> <p>1. Form: Symmetry and Distribution.</p> <p>2. Secondary Degeneration.</p> <p>(6) CONCLUSIONS :</p> <p>1. General Features of the Areas in Case I.</p> <p>2. Their Topographical Distribution.</p> <p>3. Note on the Pathological Physiology.</p> |
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(1) INTRODUCTION.

It has already been briefly mentioned that this study is based upon the observations made in the histological investigation of nine cases of disseminated sclerosis. The brain and spinal cord from most of these cases were equally thoroughly examined, but in only one instance was there the possibility of following the case clinically and anatomically. That case, therefore, has been taken, more or less, as a standard of comparison, and it is proposed to begin this part of the paper by giving a short account of the clinical history of this patient.

Clinical History.

L. W., aged 28, a kitchenmaid by occupation, was admitted to the late Dr Alexander Bruce's wards on 4th April 1910, complaining of weakness in both legs, inability to walk, and tremors in both arms and legs, the duration of these symptoms being about two years.

History of Present Illness.—At the end of March 1908, the patient, who was then employed at one of the baths in Edinburgh, was reaching up to clean the wall with a long brush, when she stepped too far back and fell into the deep end of the bath. She was pulled out immediately, but was, of course, wet through to the skin. This gave her a great fright, and she screamed for about ten minutes. When she had quietened down, she had a hot bath, put on dry clothes,

and went home. She said also that a menstrual period came on about two hours before she fell into the water; after this it completely stopped, and did not return until a fortnight later. The above accident took place in the forenoon, and by the afternoon she felt so much better that she returned to her work.

She continued at her work until the end of June, when one morning she found that both her legs had become swollen and tender, that she had shooting pains down her right leg, and that she had great trouble in rising out of bed. She sent for her doctor, who thought the symptoms were due to muscular rheumatism, and treated her accordingly. She was slightly feverish at this time and remained in bed for a week, and at the end of a fortnight returned to work. She noticed, however, that she did not seem to be able to walk so quickly as previously, that she was slightly lame, and that her right knee was stiff.

She worked steadily at the baths until March 1909, when she began to find her work too heavy, and she, accordingly, took an easier situation as kitchenmaid at a golf club-house. She succeeded so well at this that after a month she became waitress. After four months this post became too heavy for her, and she returned to her old post of kitchenmaid, but two months later had to give this up also, and has done no work since 12th November. Two months before admission her symptoms had become more marked. Both legs became very stiff, and she could only walk with the help of a stick. Her arms became shaky, and one of the reasons why she left the club-house was that if anyone gave her a sudden start her arms became very shaky, and once or twice she dropped a tray full of dishes. Although usually constipated, she was subject to sudden precipitate action of the bowels as well as to incontinence. Her friends had noticed that she had been getting very slow in her speech, and that sometimes she had difficulty in pronouncing words. The menstrual periods had been somewhat irregular, usually occurring too frequently, but lasting only a very short time. As these symptoms became progressively worse, she was sent to the Royal Infirmary, Edinburgh, to be examined by the late Dr Alexander Bruce, who at once admitted her to his ward.

Previous Illnesses.—Patient had always been remarkably healthy as a girl, and, with the exception of occasional "bilious headaches," could remember of no other illnesses.

Condition on Admission.—Her appearance was that of a strong healthy girl with a good fresh colour. Smell and taste were normal. The pupils were circular and equal, and reacted both to light and to accommodation. The eyes were freely movable in all directions. There was slight lateral nystagmus on looking to the left and slight rotatory nystagmus on looking upwards. Both discs were normal.

Sensation to touch, heat and cold, and pain was intact; and there was some tenderness on deep pressure on both calves.

The knee jerks were both exaggerated, the left rather more than the right. Both Achilles jerks were also exaggerated, and Babinski's

sign was positive on both sides. The abdominal and upper limb reflexes were all present and apparently normal. There was no wasting of any muscles. There was slight inco-ordination in the finger-nose test with the right hand, but no intention tremor and no inco-ordination of the lower limbs.

The heart was not enlarged, the apex-beat not displaced, and the pulse was 70. There were no murmurs present, and the arteries were not thickened nor the blood pressure raised.

The chest was well formed and the expansion good. Neither percussion nor auscultation revealed anything abnormal.

There was no enlargement of the liver or spleen. The stomach was not dilated, the digestion was good, but there was considerable constipation. The urine contained neither albumen, sugar, bile, nor pus, the specific gravity being 1,017. She has occasional attacks of precipitate micturition.

Progress.—She was put to bed, and at first made good progress, but on 25th May numbness in the left arm developed suddenly. On the 29th she felt deaf in the right ear, with dull buzzing sounds, and noticed that her right eye watered exceedingly. She complained of feeling her face squint on speaking. On examination a right facial paralysis was found. The right eyelid scarcely moved on attempting to close the eye. The electrical reactions on the affected side of the face were normal both to faradism and to galvanic stimulation. On the 3rd of June she developed diplopia on looking to the right, and was found to have a paralysis of her right external rectus. The other movements of the right eye were normal, and the left eye was unaffected. The pupils were equal, rounded, and dilated, and reacted to light and to accommodation sharply. Five days later the tongue was seen to protrude to the left side, and slight difficulty in speech was observed, which was followed two days later by difficulty in swallowing. Dr J. S. Fraser, who at this time examined the ears, reported that both vestibular apparatuses were functional, although possibly the activity of the right was slightly diminished. By the 8th of August the weakness in the legs had become very marked, and they were painful on attempting any movement. Spasticity in the legs soon developed. On 14th August severe vomiting set in, which was scarcely affected by any treatment. On the evening of 15th August she complained of dimness in seeing, and next morning was found to be blind in both eyes. On examination the discs showed some pallor, but no neuritis. Muscular wasting now developed and proceeded rapidly, especially in the legs. The face became more hollow and sunken, diarrhoea and vomiting set in, and a catheter had to be passed every eight hours. From now onwards she went rapidly downhill, sometimes slightly better and sometimes slightly worse. She had an extremely septicæmic look, and died suddenly and unexpectedly on the morning of the 5th of September, after having passed a better night and had a little breakfast.

Post-Mortem Report.

Body is small, well developed, and fairly well nourished. Rigor passing off in upper limbs and in lower limbs. Slight amount of œdema of lower limbs. Pupils are slightly contracted and equal.

Serous Sacs.—No adhesions and no fluid in pleural cavities. No excess of fluid in pericardium.

Heart.—230 grams. Rather small. Shows a few small milk spots on anterior aspect of ventricles. Tricuspid valve at C.C., 12 cm. Appears healthy. Pulmonary valve competent. C.C., 9 cm. No pathological change. No dilatation of either ventricle. No sub-epicardial fat. No thrombi in cavities. No post-mortem clot. Mitral valve C.C., 11 cm. Healthy in appearance. Heart muscle rather pale but firm. Coronaries healthy. Aorta and aortic valves also healthy. Aortic orifice C.C., 8 cm.

Lungs.—Lungs are voluminous and pale. General atrophic emphysema, especially along anterior border. Adhesions between upper and lower lobe in left lung. Collapse in lower lobe. On section the lung shows œdema in both upper and lower lobes. Bronchi contain a good deal of frothy mucus. No special change in their walls. Right lung adhesions between upper and middle lobes. There is a stoney hard nodule in lower part of the upper lobe. On section the lung is œdematous and has slight congestion of lower lobe. Left lung, 290 grams. Right lung, 400 grams.

Liver.—1,470 grams. Normal in size. Gall-bladder is very small, and contains a small quantity of brownish-red bile. No evidence of gall stones. On section liver substance is pale in colour, rather friable and soft. There is some diffuse fatty change, slight in extent.

Kidneys.—Left, 220 grams. Left suprarenal gland is large. No special change. On section, kidney slightly enlarged. Cortex as a whole is diminished in breadth. It is pale. At upper end there are several opaque white areas with hæmorrhage around them. General dilatation of pelvis. Capsule strips easily and leaves smooth lobulated surface (fœtal). Right kidney, 200 grams. Right suprarenal is also large, but shows no special change. Right kidney distinctly enlarged. On section shows numerous abscesses. Pelvis dilated and shows pyelitis.

Other Abdominal Organs.—Spleen, 75 grams. Not abnormal in size. Pale colour and soft in consistence. Hæmorrhage into substance. Stomach dilated. Mucous membrane pale. Covered with glairy mucus. Shows minute hæmorrhages. Some chronic gastritis. Early marked dilatation of veins in lower part of œsophagus. Mucous membrane of duodenum is somewhat congested and shows a few small hæmorrhages. Pancreas is pale and appears healthy. Small and large intestines show nothing abnormal in mucous membrane. Bladder shows some thickening: interior grey and necrotic in places. Uterus and appendages appear normal.

Brain.—No clots in superior sinus. Convolutions atrophied. Some general opacity of pia arachnoid. Fluid in subarachnoid space.

Cord.—Shows irregularly scattered areas of bluish-grey colour varying in size and shape. Cord as a whole is small. Similar areas in pons and medulla.

Macroscopically the cerebral and spinal meninges were in the main normal, but in places where the grey and greyish-blue areas in the cord reached the surface the pia over these areas appeared slightly opaque. On section of the cord at various levels, numerous gelatinous grey areas were found, and also parts of a softer consistence with a whiter colour than the normal tissue of the cord. Sections of the medulla oblongata and pons indicated the same two types of areas, parts of the pons appearing so affected that there were only islands of normal tissue, and the floor and immediate neighbourhood of the fourth ventricle were also markedly involved. Numerous areas were found in the horizontal sections of the cerebral hemispheres at various levels, not only in the basal ganglia and white matter, but also in the cortex and subcortical white matter. There was a well-marked periventricular sclerosis, especially of both the posterior horns of the lateral ventricle extending down into the descending horn on both sides. The ventricles were not dilated, and their surface was smooth but cloudy, and raised in slight ridges, corresponding to the venous branches which are present underneath the ependyma, especially at the posterior and anterior horns of the lateral ventricle. These veins were distinctly outlined and surrounded by a zone of gelatinous tissue.

The size of the areas varied very considerably: in the cord it was impossible to define macroscopically, either on the surface or on section of any of the areas, as they seemed to run into one another. In the brain, however, isolated areas were the rule, though here also irregular areas of different size and form coalesced. Those isolated in the white matter varied in diameter from 1 to 10 or 12 millimetres, and were mostly oval or circular. The largest were found in the immediate neighbourhood of the roof of the lateral ventricle, and gave the impression of being upward extensions of the peri-ventricular sclerosis of the roof.

In examining the microscopic findings it is important to recognise that different stages of the disease come under observation, and it would appear natural to differentiate acute, subacute, and chronic stages, for they are often enough found close together in one and the same case. The finding at the autopsy of patches at different stages in their evolution accords with the clinical history of the affection, which has usually progressed with remissions and relapses. It is, however, by no means universally admitted that the recent areas are transformed into those which would seem to represent a chronic process, in which all traces of inflammation have disappeared. If such chronic areas do not develop on the basis of the former, it must be acknowledged that

there is a strong justification for looking upon them as a multiple gliosis, related to an anomaly of development, and upon the recent areas as an acquired form of sclerosis, whose origin lies probably in toxi-infective conditions.

This study, therefore, begins with a short description of these two types of areas; the one an old area, typical of the "sclérose en plaques" of the earlier writers; the other, a recent area, corresponding to those found in the so-called acute multiple sclerosis of recent writers. It will then be necessary to endeavour to trace all the stages in the development of an actual sclerotic area, and further to describe other types of areas present. A second section will deal with the structure of areas in special situations, *e.g.*, peri-ventricular sclerosis, cortical and combined subcortical and cortical areas, areas in the grey matter of the cord, areas in the cerebellum, nerve roots, &c. A further section will deal with the changes in the individual structural components of the central nervous system, and a final section with other features, *e.g.*, the form, symmetry, and distribution of the areas, and changes, such as secondary degeneration, outside of the area.

(2) STRUCTURE OF DIFFERENT TYPES OF AREAS.

1. *An Actual Sclerotic Area.*

(a) *In the Spinal Cord.*

- (i) Nerve fibres cut longitudinally, *e.g.*, in the ventral third of the posterior columns (Figs. 4, 31, 161-166, and 171, 172).

The histological structure of such an area is a very simple one. It consists almost entirely of newly-formed fibrils which are arranged parallel to the original course of the nerve fibres. In sections stained by means of Weigert's medullated sheath stain and counterstained with picro-fuchsin,¹ under a low magnification we recognise at once three of the outstanding characteristics of an old sclerotic area (Fig. 165): the disappearance of the myelin sheath of the nerve fibre, the proliferation of the glia, and the alterations of the blood-vessels. By means of a glia stain (Fig. 163) and a diffuse stain (Fig. 166), these changes are confirmed

¹ Such sections will in future be designated Weigert sections.

and their details revealed; by an axis cylinder stain the fourth histological characteristic is represented—the persistence of numerous axis cylinders (Figs. 164, 254); and, by the Marchi method, it is seen that this old sclerotic area contains no degenerating nerve fibres nor granular cells containing the products of the degenerated myelin.

The shape of this area is brought out clearly by Weigert's stain as an elongated oval, with its long diameter in the long axis of the cord (Fig. 31). The continuity, therefore, of the nerve fibres is broken by this oval area, and the absence of the myelin at the sides is more or less sharply defined from the surrounding tissue, the margins forming more or less sinuous lines, but the limits of the area, on longitudinal section, are never straight or even curved outlines, for individual nerve fibres or bundles of such pass into the borders. The myelin sheath of these fibres stains irregularly and weakly, and the termination is usually broadly broken off, or it may be swollen or narrowed (Fig. 236).

Numerous fine glia fibrils course in regular, undulating lines parallel to and close to one another (Figs. 3, 164). The neuroglia nuclei lie often in short rows or in groups, but more often, as in this area, isolated with the long diameter in the long axis of the fibrils, and, as a rule, quite independent of them. The number of the nuclei is smaller than in the normal tissue, but in structure and form they are slightly larger, clearer, and more oval. Between the glia fibrils and surrounded by them are found numerous axis cylinders, which are mostly thin and fine, but thicker and more homogeneous in structure than the glia fibrils (Fig. 164). They do not run in such regular lines, and frequently show as faint diffusely-stained bands. The impression is often given that in place of the myelin sheath there has been a substitution of glia fibrils, forming a glious sheath to the axis cylinder. Lapinsky has wrongly interpreted this process of substitution as a metamorphosis of the medullated sheath—a conception which accentuates the enveloping character of the glia fibril proliferation.

The blood-vessels stand out clearly in this sclerotic tissue. Those cut transversely show, with picro-fuchsin stain, thickened, homogeneous, pink-stained walls, and on longitudinal section the numerous longitudinally-running small vessels (Fig. 172) have a similar structure, with few cell elements even in their adventitia. The lumen of the smallest capillaries is often obliterated and that of

the larger vessels narrowed. Weigert picro-fuchsin sections show beautifully the arrangement and distribution of these smaller vessels, and bring out the abundant vascular supply of the various tissues.

At the periphery of the area, especially at its upper and lower limits, diffuse stains bring out a zone where intensely stained nuclei are in great abundance: these nuclei, in iron-hæmatoxylin stain, are seen to occupy the spaces between the nerve fibres projecting into the sclerotic tissue, and their increase can be traced for a small distance among the fibres of the normal tissue on all sides. The glia fibril proliferation and thickening of the vessels are here also evident, and this zone can be looked upon as a zone of transition between normal and sclerotic tissue—a zone where the pathological changes gradually cease till normal tissue is reached.

- (ii) Similar area in the cord with nerve fibres cut transversely (Figs. 88, 89, 183, 184, 188-190).

To understand the structure of such an area it must be remembered that in the posterior columns, according to Weigert, the usual glia fibrils very largely run longitudinally, and the pathological glia fibril formation takes place almost wholly in this direction. Exceptions to such an arrangement will be noted later. When the glia proliferation has taken the simple course represented in the former paragraphs, a transverse section of the areas in Figs. 88, 89 will represent the glia fibrils as minute fine points which more or less surround as a ring larger, more diffusely stained, and more homogeneous points—the axis cylinders. If the section is directly transverse to the direction of the fibres we get so dense a picture that, under low power, no details can be recognised (Fig. 184). But a higher magnification shows that the normal meshes of the glia and the space originally occupied by the myelin sheath of each nerve fibre is replaced by those fine points. Amongst them, lying almost isolated and in no way forming the nodal points of a reticulum, are rounded nuclei, with smooth nuclear membrane and clearer nuclear structure than the small normal glia nuclei. In this dense tissue is found the cross-section of numerous thickened capillaries and pre-capillary vessels (Fig. 273). Around each vessel is a narrow zone where the glia fibrils radiate almost perpendicularly to the vessel wall—forming the “corona

ciliaris" described by Borst and Storch. It is not a question of a central vessel, but of many transverse and oblique vessels. These give the impression that not one single vessel, but the branches of a vessel system, are affected. Weigert sections show the complete absence of myelinated fibres within the sclerotic tissue, but at the periphery the transition to normal tissue is a gradual one— isolated well-stained myelin sheaths being found within definitely sclerotic tissue (Fig. 88). The transition zone is again seen to consist of a large number of small deeply-stained nuclei, amongst which are found larger nuclei with a distinct amount of protoplasm and several protoplasmic processes (Fig. 187). Here a more reticular arrangement of the glia fibrils can be recognised: the glia trabeculae which, in the normal tissue, separate groups of nerve fibres, are in this transition zone more evident, and the processes of the cells cut into the larger meshes, dividing up the bundles into smaller and smaller groups, till, finally, the meshes contain only individual nerve fibres, and the granular appearance of the sclerotic area is reached. Sections of such an area stained by the Marchi method again show no black staining: the characteristic transverse section of the myelinated fibre is absent, but no traces of degenerating myelin are found either in the fine tissue spaces or around the blood-vessels.

The impression is received that here again the normal architecture of the tissue is preserved; that the myelin sheaths have disappeared (Fig. 88) (Weigert stain), and have been replaced by a fine close fibre formation (Fig. 184) (glia stain); that the walls of the blood-vessels, even the small capillaries, have thickened walls with few cell elements (Fig. 270) (diffuse stains); that the axis cylinders have, at least to a considerable extent, been retained (Fig. 257) (silver impregnation stains); that there are at present in the area or its periphery no indications of the degeneration of myelin (Marchi method)—thus allowing it to be supposed that the process which caused the myelin destruction has come to a standstill; and, finally, that there are evidences at the periphery of the area, in a nuclear proliferation and diminution in myelin fibres, of a process which has left these traces of a reaction to its further progress.

These areas, just described, in longitudinal and transverse section, are typical of the sclerosis which is the essential substratum of the disease known as disseminated sclerosis. They show a compact glia fibril proliferation, apparently without spaces,

for only here and there are there traces of the original glia meshes, The area of dense sclerosis is regularly surrounded by a peripheral transition zone, which interposes between it and the healthy tissue. This transition zone shows very numerous nuclei of small and large glia cells, with more or less developed glia trabeculae.

(b) *In the Cerebral White Matter.*

The first points to be noted in comparing such an area with a similar one in the spinal cord are that in the brain it is much more usual to get a more defined outline of the patch, and also that a central vessel, cut transversely or longitudinally, is more frequently present.

- (i) Nerve fibres cut longitudinally, *e.g.*, at the base of or within a medullary ray (Figs. 118, 202).

The shape of the area is again often oval, and its structure is very similar to that described in the longitudinal direction of the cord. Weigert sections show again a complete absence of myelin (Fig. 114); the presence often of one thickened vessel, which sometimes is found to extend centrally almost the whole distance of the patch (Fig. 117); and the presence also of numerous other smaller vessels cut transversely, obliquely, and longitudinally—all with walls thickened, almost homogeneous and structureless. The general substance of the area is again composed in great part of fine glia fibrils (Fig. 202), which run longitudinally and parallel to each other, and surround the persisting axis cylinders (Fig. 252). The glia cells in this area are, as a rule, much less numerous than normal; they have, however, more elongated and lighter-staining nuclei. Along with these many darkly-staining smaller nuclei are found, which agree in form and staining with the normal small glia cells of the cerebral white matter. The preserved axis cylinders are, as a rule, much fewer in number than in the cord, and they show, even in the dense sclerosis, more evident indications of a previous involvement. They are usually thicker, more diffusely stained, and have more irregular contours as compared with the axis cylinders of adjoining medullary rays. Marchi-stained preparations again show a complete absence of any degenerating myelin either in nerve fibre or in the presence of fat granule cells. The affected area stands out much lighter in colour than the surrounding tissue, and gives a contrast almost as marked as the

negative picture of the Weigert section. In glia sections this condensed tissue is, on the other hand, much denser and darker than the surrounding tissue, and its limits are often very defined—that towards the central white matter being abrupt, that towards the radiations of the convolutions being very frequently in the form of a wedge with its apex to the radiations (*cf.* Fig. 235). The transition zone at this limit is also much more marked, and consists of a narrower or broader zone of deeply-staining round nuclei, amongst which a few larger, protoplasmic forms are found. This nucleated zone extends for a short distance into the normal myelinated tissue.

- (ii) When the sclerosis affects a portion of the brain substance in which the nerve fibres normally run in very varied directions (Figs. 122, 123; 194; 227 228; 260), *e.g.*, a small defined area in the central white matter above the roof of the lateral ventricle, the resulting tissue is a very dense network in which the original glia spaces are replaced by fine fibrils.

On Weigert picro-fuchsin sections the central vessel stands out clearly as the mid-point of a yellow-stained zone, at the periphery of which there is usually an abrupt transition to normal myelinated tissue (Fig. 124). If bundles of longitudinal fibres are cut across, these often pass for a short distance into the sclerotic zone, breaking the otherwise almost defined circular or oval outline of the area (Fig. 122). At their terminations such fibres do not show any evidence of degeneration, but are simply faintly-staining normal fibres. In this sclerotic zone there are found as a rule, in addition to the central thickened vessel, several cross-sections of capillaries, each standing out as a pink thickened ring, which encloses a narrow lumen. Individual capillaries are quite obliterated and form a dense solid fibrous cord. Marchi-stained preparations again show a complete absence of signs of disintegration of myelin: the affected area again appearing lighter in colour than the normal tissue.

Sections stained for glia and for cell structure show that the sclerotic tissue is composed almost entirely of glia fibrils. These fibrils are very unequal in size: the larger form larger meshes into which the finer fibrils cut and make finer meshes. The tissue becomes denser and denser till the meshes are inconceivably

fine (Fig. 194). The size of the glia cells varies very much, but the body of the cell is in general round or slightly elongated and may contain one or more nuclei, but little protoplasm. Around the blood-vessels (Fig. 228), even the capillaries, is a zone in which this fibril formation is even more dense; the fibrils rarely form a "corona ciliaris," but rather concentric layers of fibrils closely pressed together, with few nuclei, form an outer dense glious sheath to the vessel. The axis cylinder content of these areas has been exceedingly difficult to ascertain, for the brains of most of the cases in which such areas occurred were already fixed in formalin, and the Bielschowsky impregnation method in cerebral areas gave no absolutely reliable results. A comparison with other areas, stained by means of Cajal's silver method, showed how extremely difficult it was to differentiate between glia fibrils and fine axis cylinders and their branches. The intimate network formed by both is so alike that it was never possible to be satisfied that in these dense sclerotic cerebral areas there could be so abundant an axis cylinder network persisting, and the conclusion was come to that in such areas the glia network had been stained. Other areas which had been cut in two by the section of the hemispheres were taken through—one part by Cajal's method, and one for glia and cell staining. These showed that when the fibrillar network of the glia was not quite so dense as above described, there was a very abundant network of axis cylinders and their finest branches persisting (Fig. 260). Such areas will be referred to in a later section.

The nucleated transition zone of these dense sclerotic areas was, as a rule, a narrow one, and the nuclei were all relatively small and darkly staining (Fig. 233). Even in the sections stained with diffuse stains the contrast between the sclerotic tissue and the normal was very evident.

Such areas, therefore, consist of a dense-meshed fibrillar tissue, poor in nuclei, with thickened vessels. The areas give the impression again of tissue in which the normal architecture is retained: they also seem to show that the change consists in a demyelination, a complete substitution of the spaces by fibrillar tissue—which in its arrangement forms simply a condensation of the original glia meshwork (Fig. 194), a thickening of the walls of the vessels originally present in the tissue (Fig. 270), and, finally, the formation of a narrow but dense nucleated peripheral zone (Fig. 233), which forms the transition to normal tissue.

2. An "Early" Area.

(a) *In the Spinal Cord.*

- (i) Nerve fibres cut transversely (Figs. 10; 90, 91; 66, 143; 180), *e.g.*, in the middle or anterior third of the posterior columns.

On Weigert sections an irregular non-medullated area can be recognised (Fig. 90), which extends approximately from the middle of the posterior median septum forwards to the posterior commissure. The area is, roughly speaking, triangular in shape, with its base to the commissure, and under low power has a more or less definite sinuous outline. At the periphery on all sides there are isolated nerve fibres passing for a short distance into the area, and under high power it is seen that their myelin ring is very thin, or stains diffusely, and shows evident signs of degeneration. This area has formed with the posterior median septum and its vessels as a centre (Fig. 143), and on either side are found numerous cross-sections of small vessels and capillaries and longitudinally-running branches of the posterior median fissure vessels. Individual branches of these longitudinal vessels can be followed up into the surrounding normal tissue. The tissue of this area, even in Weigert sections, is seen, notwithstanding the absence of myelin and the presence of changed vessels, not to correspond to the dense sclerotic fibrillar tissue seen in the former area described. The blood-vessels—arteries, veins, and capillaries—are all dilated and engorged with blood, and their walls, instead of the homogeneous, thickened, structureless tissue, show in the intima and media little recognisable change from normal, but the adventitia has its lymphatic spaces dilated and filled with nucleated elements, and these same nucleated elements are scattered irregularly through the non-myelinated tissue, and in the blood-vessel walls of the transition zone (Fig. 10).

The most characteristic features of this area are seen best with diffuse stains. Hæmatoxylin and eosin, or Van Gieson's stain, or, even better, Heidenhain's iron-hæmatoxylin stain, show the presence of a very large number of nucleated cell elements with a considerable amount of protoplasm. Most of these large cell elements correspond roughly to pathological examples of the proliferated spider cells of the normal tissue (Fig. 9). The nucleus is large, vesicular, with a very light chromatin framework, and one

or more distinct nucleoli. It lies usually excentrically in the protoplasm: this is large in amount, usually homogeneous, and stains slightly with hæmatoxylin or yellowish-green with the picro-fuchsin. From the protoplasm radiate in all directions very fine branching, protoplasmic processes, which break up into a fine network. Many of these cells are multi-nucleated, and vary greatly in shape and size from star-shaped forms to those with crescentic outline and bi-polar forms. Their processes bear very frequently a very definite relationship to the vessel walls—a relationship which will be more fully emphasised when describing recent areas in the cerebral white matter. Smaller and darker stained nuclei, with little protoplasm and no processes, can also be found in considerable numbers. In the spaces formed by the large branching processes of these cells and in the adventitial spaces of the blood-vessels lie the second tissue elements characteristic of such an area. These are larger, rounded, nucleated cells, with a large amount of vacuolated protoplasm: they correspond to the phagocytic cell of the central nervous system, and their vacuolated appearance is derived from the solution of the fine degenerated myelin granules in the process of hardening. Nissl has given to these cells the name "Gitterzellen" from their morphological appearance, but they are more generally known as compound granular or fat granular cells ("Fettkörnchenzellen") (Figs. 9, 10), from their function of absorbing the products of the disintegration of the myelin. These cells lie not only in all the tissue interstices, surrounded by the branching processes of the glia cells, but in the lymphatic spaces of the adventitia of the blood-vessels—whither they have in all probability been drawn in from the tissue spaces by the suction influence of the lymph flow in the adventitial lymphatics. Round even the smallest capillaries these cells form a complete ring, and the area under low power assumes a very characteristic appearance. The presence of such large numbers of fat granular cells gives to the blood-vessel walls an appearance of a cell-infiltration, for in the vessels larger than the capillaries they are present in several rows, often closely compressed. In addition to these cell elements in the adventitia, other nucleated elements add to the nuclear abundance of the vessel wall. The endothelium, especially of the capillaries, frequently shows evidence of a distinct proliferation, and in the adventitia are found dark-stained nuclei, together with nuclei of a vesicular clearer

character, both of which have probably arisen from the proliferation of the cells of the adventitia and the endothelial cells of its lymphatic spaces. Diffuse stains, *e.g.*, Van Gieson's stain, bring out, although feebly, one further tissue component—the axis cylinders. These are no longer the sharply defined, homogeneous points of the dense sclerotic tissue, but a faintly-staining, almost unrecognisable, swollen structure, which is a lesser degree of the intense swelling of the axis cylinder seen in marked oedema of the cord. These swollen axis cylinders are usually found lying in the tissue meshes, with no trace, or only slight traces, of myelin around them, and often closely compressed between the fat granular cells and the protoplasmic processes of the large glia cells (Fig. 10). The iron-haematoxylin stain shows that in this area there is as yet almost no attempt at fibril formation on the part of these glia cells, although the processes at their lateral margin and terminations show a definitely darker staining.

It has been seen that Weigert sections show the almost complete absence of myelin in this area; that diffuse stains give (1) the characteristic appearance of the two cell elements—proliferated glia cells with numerous processes and the fat granular cells, (2) the numerous dilated blood-vessels, and (3) the persistence of numerous swollen axis cylinders; and that glia stains give as yet no definite fibril formation. There remains now to be mentioned the appearance with Marchi-stained sections. This is the most characteristic of all, and has given to the areas the name of “fat granule cell myelitis.” Each of the large vacuolated cells in the tissue spaces and walls of the blood-vessels is found to be composed of a very large number of minute granules of a substance staining black with osmic acid. Most of these granules are quite round, but a few show irregular contours from compression. Granules are found also in the spaces between the cells. Under low power the area is thickly studded with these black granular cells, which also form concentric rings around the blood-vessels. No trace of normal myelinated fibres can be found within the area, but in the transition zone are recognised numerous fibres in all stages of degeneration. The blood-vessels in this zone also have their sheaths filled with similar cells, but the numbers in the tissue spaces themselves are still too many to allow the radial appearance to be recognised, which is characteristic of the areas in which the blood-vessels

radiating from the area have their walls filled with cells which have passed from the tissue spaces to the lymphatic spaces of these vessels, leaving the tissue more or less clear. In this transition zone, in addition to numerous degenerating myelin fibres and vessels with rows of fat granular cells in their walls, we find also a marked proliferation of the glia cells and protoplasmic processes, and a widening of the normal glia meshes, but the degeneration of the myelin has not advanced to the stage of complete disintegration and its absorption by cells. The glia meshes of the adjoining normally myelinated tissue are distinctly widened, the cells also enlarged, and the blood-vessels engorged and dilated.

(ii) Similar "early" area in the cord—nerve fibres cut longitudinally (Figs. 1, 2; 18, 20; 156, 168).

In Weigert sections under low power this area is both laterally and at its upper and lower limits very irregularly defined. The transition zone on all sides shows that the fibres, though still staining with hæmatoxylin, are markedly altered (Fig. 236). Longitudinal sections are very valuable in showing the changes in the nerve fibre as it passes into the area. Those changes will be referred to in detail in a later section. In the area itself are numerous globular and granular remains of the myelin which have not undergone complete disintegration into fat droplets (Fig. 237), and one can also recognise, even within cell elements, such remains which still retain the myelin stain.

Marchi preparations (Figs. 146-148) show the characteristic appearance of long rows of fat granule cells, which seem to occupy the spaces left by the removal of the degenerated myelin of the nerve fibre and also the longitudinally-running vessels surrounded by elongated layers of similar cells (Fig. 263). In sections stained with Scharlach R. and hæmatoxylin (Figs. 18-20), the smallest capillaries can be followed, marked out by a single or double row of such cells, the nuclei of which is either central or pushed to the periphery by the accumulating granules of fat. In the transition zones of the area the disintegration of the projecting nerve fibres can be followed and the formation of the fat granule cells, which can be traced not only into the transition zone, but even between the nerve fibres of the normal tissue.

In sections stained with diffuse stains, the large glia elements,

already referred to, are beautifully seen, lying often in rows (Figs. 167, 209), with the nuclei of two adjoining cells lying close to one another, as if they had arisen from the division of the cell. The long branching protoplasmic processes extend round and almost envelope the rows of fat granule cells, and in some parts the direction of these processes is already becoming longitudinal. Almost pushed aside between the rows of cells and the protoplasmic processes can be found faintly-stained, homogeneous, swollen bands, which represent axis cylinders (Figs. 1, 2), and here and there are large numbers of rows of finer and larger granules staining with the same tone as these bands. These are the remains of disintegrated axis cylinders, which seem, after the stage of severe swelling, to become broken up into large granule formations, and gradually into finer granules before they ultimately disappear (Figs. 255, 256). These remains of axis cylinders can be distinctly recognised not only with eosin, picro-fuchsin, and iron-haematoxylin, but also with the elective axis cylinder stains, *e.g.*, Cajal's and Bielschowsky's methods.

(b) *In the Cerebral White Matter.*

- (i) Nerve fibres cut longitudinally (Figs. 114, 134, 200, 203-206), *e.g.*, an oval area at the base of or within a medullary ray.

Such an area is again very similar in structure to that described in the spinal cord. Weigert sections show that there is a gradual transition on all sides into healthy tissue (Fig. 114), and that the adjoining nerve fibres show marked traces of swelling, diffuse staining, and granular disintegration. The blood-vessels appear very numerous, and even the capillaries are dilated, engorged with blood, and, in nuclear staining, show an evident increase of the endothelial nuclei, together with a very abundant presence of fat granular cells in their adventitial spaces. The two most characteristic features of the cord area are here again present: (1) the rows of fat granule cells, which seem to occupy the tubular spaces of the degenerated myelin fibres (Fig. 203); and (2) the presence of rows of large, protoplasmic glia cells (Fig. 205) whose processes entwine between the rows of fat granule cells and separate them into more definite layers. These processes divide into numerous fine interlacing and anastomosing branches

which tend to take longitudinal direction, the protoplasm of some of the cells being drawn out also in a longitudinal direction and giving off numerous processes from either pole. The glia nuclei are vesicular and elongated, and frequently one or more are at either pole of a cell. Together with the larger nuclei are found many smaller, darker-stained nuclei, with little protoplasm and no processes.

The adventitial wall of the longitudinally-running vessels is again infiltrated with fat granule cells, which form a single or double layer, and when the capillaries join the smaller vessels the whole tissue in the angles between the vessels seems permeated with such cells. In the walls of the vessels are found other nucleated elements which are rounder, denser, and have only a small cell body, and similar cells are found frequently in small groups or isolated in the tissue immediately adjoining the capillaries. The axis cylinders are here very thickened and vesicular, and in many parts transformed into clumps of larger and smaller globules and granules. In all cases they seem pushed aside by the large glia cells and rows of fat granule cells, and take the direction between these rows. Marchi-stained preparations (Figs. 134-136) bring out very beautifully the enormous number of fat granule cells and the changes in the nerve fibres as they pass into the affected area. The transition zone is a very broad and irregular one, and can be more clearly understood from transverse sections of such an area.

Such an area, therefore, consists of elongated rows of fat granule cells, almost alternating with rows of large, protoplasmic glia cells with long-branching processes. In between the rows are numerous capillaries and larger vessels, cut mostly longitudinally, and surrounded by one or more layers of fat granule cells. Numerous axis cylinders have perished, and those persisting are markedly altered. There is a complete absence of myelin within the area, and the transition into normal tissues is a very gradual one.

- (ii) Small "early" area in cerebral white matter, with nerve fibres cut in various directions (Figs. 5, 6; 68; 191, 192).

The most important and characteristic feature of such an area is the enormous number of large cell elements. These are of two

kinds: the one the round vacuolated fat granule cell with central or peripherally-placed nucleus (Fig. 195); the other the large protoplasmic glia cell with branching processes (Fig. 6). Both of these cell elements are seen here at their most distinctive stage of development. This area (Fig. 5), situated in the central white matter at the base of one of the parietal convolutions, measured less than 2 millimetres in diameter. It was almost circular, and was surrounded by a well-marked nucleated transitional zone. In Weigert sections one saw a well-marked central engorged vessel with an increase of nucleated elements in its walls, a complete absence of myelin within the area, and the presence of numerous rounded spaces, almost equal in size, which gave the non-myelinated tissue a very characteristic fenestrated appearance. With cell stains it was recognised that these spaces are fat granule cells, so closely arranged together that one might almost think of them as forming irregular tubular lines made up of cell units—the tubules almost as closely arranged as the rows of liver cells in a lobule. Between groups of these cells lie large glia cells such as are nowhere found in the normal glial tissue. These are large cells with homogeneous cell body: their size sometimes as large as that of a motor ganglion cell of the cord. They have large-branching, protoplasmic processes which wind around the individual fat granule cells and almost surround and isolate them. Their nucleus is large and vesicular, and contains a very defined membrane and one or more deeply-stained nucleoli—its position is usually excentric, but is sometimes central. These cells differ from the Deiters or spider cells only in degree, and are pathological spider cells, and they may be multi-nucleated. They are found specially numerous in the neighbourhood of the small blood-vessels, and their processes are frequently attached to the adventitial wall (*cf.* Figs. 265, 266). With glia stains it is seen that the margins of the processes of these cells are already becoming differentiated into fibrils (Fig. 212) which are found lying between and surrounding the fat granule cells. The central vessel is dilated and engorged with blood, and in its adventitia is an increase of nucleated elements. These are mostly small, round, deeply-staining nuclei, with little protoplasm, together with a few fat granule cells. The capillaries within the area are also dilated, but changes in their walls are not marked. The presence of swollen axis cylinders in cross-section and in short pieces in

longitudinal can be proved in the narrow spaces between the fat granule cells and the glia cell processes.

At the periphery is a more or less broad transition zone. This shows (1) a very irregular loss of myelin, the myelinated fibres showing all stages of degeneration; (2) a very marked nuclear proliferation, which in the outer part consists mostly of small nuclei and in the inner part of the larger, protoplasmic cells similar to those just described; and (3) the presence of a few fat granule cells lying in the wide meshes between the processes of the large glia elements.

Marchi sections of such a small recent area (Figs. 13, 68) show the central vessel surrounded by concentric layers of cells filled with closely-compressed fat granules. Similar cells lie irregularly but closely scattered in the tissue, and the picture enables one to recognise how such areas received the designation of "fat granule cell myelitis." Marchi sections counter-stained with safranin and mounted in Canada balsam give a very instructive picture. The fat granules within the cell elements dissolve in the Canada balsam, leaving a skeleton structure of the tissue with the nuclei and the processes of the large glia elements and the nuclei of the fat granule cells stained with safranin. Such sections frequently gave the most characteristic representation of the close relation of glia cell processes to the fat granule cell—the latter apparently lying in the large meshes formed by the processes of different glia cells. The Marchi preparations also showed that the fibres in the transition zone were in a condition of disintegration. This degenerating myelin did not dissolve out when mounted in Canada balsam—thus showing the different constitution of the fat granules within the cells and the globules and fragments of disintegrated myelin.

Such an area, therefore, again consists largely of closely arranged fat granule cells, between which lie the large protoplasmic proliferated glia elements; of dilated vessels with fat granule cells and other nucleated elements in their adventitial spaces; of markedly altered persisting axis cylinders; and of a gradual transition zone in which these changes are less marked and in which degenerating myelin fibres may be found.

If now the old sclerotic areas are briefly contrasted with those which have been termed recent areas, it is found that the areas typical of the "*sclérose en plaques*" of Charcot are marked by

the following histological characteristics: the complete absence of myelin (Weigert stain); the presence of a dense fibrillar tissue (glia stain); the persistence of numerous axis cylinders (silver impregnation method); the presence of numerous blood-vessels with condensed, sclerosed walls (diffuse stains); and the complete absence of any evidence of myelin degeneration (Marchi method); and, finally, a nucleated transition zone, which gives frequently an abrupt passage to normal tissue. On the other hand, the recent areas, while presenting a striking contrast to the former, have also some of these characteristics, but in a lesser or different degree: the absence of myelin within the area; a very slight commencing glia fibril formation, but a very intensive glia cell proliferation; the persistence, to a much less extent, of axis cylinders, and those persisting being markedly altered; the presence of numerous dilated blood-vessels with nucleated elements in their adventitial spaces; the marked indications of a previous myelin degeneration in the presence of fat granule cells which fill up all the interstices of the tissue and the adventitial lymph spaces of the blood-vessels; and, finally, a very gradual nucleated transition zone in which all these changes are less marked, but are combined with an evident degeneration of the myelin of the nerve fibres in this zone.

The question naturally arises: What relation do these two types of areas bear to one another? The finding of both types side by side in the same case, and the clinical history—with its remissions and relapses—argue for a close connection between them. Yet numerous writers, while not denying that the end result of the recent areas is a sclerotic tissue which bears a close agreement to the old sclerotic areas typical of the disease first designated by Charcot disseminated sclerosis, claim that true or primary disseminated sclerosis arises solely on the basis of areas of the first type, in whose evolution there is no stage corresponding to that of "fat granular cell myelitis." Areas of sclerosis which pass through the stage of the second type must be designated as secondary sclerosis, and those areas arise on the basis of a disseminated myelitis.

The question must then arise: What is the evolution of the first type? and what are the stages in the evolution of the so-called secondary sclerosis? In the next two sections an endeavour will be made to trace their respective evolution.

3. Evolution of an Actual Sclerotic Area.

A. Through Stages of Increasing Glia Hyperplasia.

The supporters of the view of a primary form of disseminated sclerosis claim that the essential lesion lies in the neuroglia tissue. By some anomaly of development this tissue, in certain areas, undergoes an intensive proliferation, which by a gradual constriction of the glia meshes produces a slow atrophy of the myelin sheath of the nerve fibre. The myelinated nerve fibre, therefore, in certain areas, undergoes a progressive reduction in its volume till it is completely replaced by the glia fibril proliferation, leaving the axis cylinder preserved. This proliferation goes a stage further than a mere substitution of glia fibrils for myelin sheath, for it is of such intensity that in no other condition is the glia proliferation so marked as in disseminated sclerosis (Weigert). It is claimed that not only is there a direct action on the nerve fibres by direct compression of the proliferating glia, but that there is an indirect action upon their nutrition. By accumulating around the vessels the glia, if it does not close their lumen, is said at least to limit their expansion, and diminish the blood-flow, and interfere with the circulation in the peri-vascular lymphatics. In this simple atrophy of the myelin sheath the degenerated products, owing to the slowness of the process, are removed just as formed, in the form of very fine granules or in solution, without requiring the presence of true granular cells. These cells are said to appear only when the process is more rapid: they may occur at the periphery of an area of true, primary sclerosis, where the affected tissue causes a reaction in the normal tissue and an excentric spread of the process occurs.

In the description of an area of old sclerosis in the cord (p. 53) an oval area in the posterior columns was chosen, because here the glia fibrils normally constitute a very uniform fine network, and the pathological increase of the fibrils appears to take place much more regularly in a direction parallel to the normal nerve fibres. In the lateral columns, however, the gradual increase of glia in a sclerosed area, on transverse section, can be followed up much more easily because of the more definitely reticular structure of the normal glia in this situation. The glia trabeculae, in the lateral column, run out transversely to the long axis, both from the marginal glia zone and from the lateral grey matter.

In the substance of the white matter they break up into a reticulum which is much coarser and more transverse than in the posterior columns, and the resultant meshes enclose the fibres and groups of fibres, which in their turn are larger than the average fibres of the posterior columns.

Fig. 173 shows an area in the lateral column, in which there is a commencing thickening of the glia trabeculae and of the finest fibres forming the reticulum. This gradually increasing thickening (Fig. 174) can be followed up till the finest glia meshes are almost obliterated and a dense fibrillar feltwork takes their place (Fig. 175). With glia and cell stains it is seen that the septa which are formed by the glia fibres around the groups of nerve fibres become thicker and denser and richer in fibrils: they, as it were, force the groups of nerve fibres more and more apart and divide them into smaller groups, and the fibrils penetrate amongst individual nerve fibrils. There is thus produced a feltwork of glia tissue, composed of glia fibrils, becoming more and more dense. As this condensation increases, even under low power it can be recognised that the ring of myelin around individual nerve fibres is becoming thinner, and finally the brilliant yellow-green ring (Van Gieson's stain) disappears altogether and leaves a naked axis cylinder. Sometimes this condensed glia has a granular, but more often a homogeneous, appearance, as if the individual fibrils had fused. Simultaneous with the thickening of the glia trabeculae and reticulum the glia nuclei in the nodes of the reticulum and within the trabeculae have enlarged and proliferated, becoming large protoplasmic cells with long-branching processes (Fig. 177). In the glia septa, large and small, in which the blood-vessels run, numerous examples of these may be found—the large ramifying processes joining the general glia reticulum. In such a condensed glia tissue the axis cylinders may be recognised for a long time (Fig. 175). They appear little altered either in quantity or quality, but may be atrophied and gradually disappear, or appear to fuse with the general glia tissue. The blood-vessels present thickened, condensed walls, with few cell elements. No compound granule cells can be found either distributed in the tissue or in the adventitial lymph spaces of the vessels. The proliferated glia cells seem to remain for a long time enlarged in this condensed tissue, and their very defined processes can frequently be traced for a long distance, with no relation to the close feltwork of the

glia in which they lie. Cross-sections of such areas are seen in Figs. 175, 177, in which the dense glia tissue appears almost fused and stains lightly with Van Gieson's stains: the numerous glia cells are still larger than the largest spider cells of the normal cord. Wiegert sections of such an area show a complete absence of myelin in the fully sclerotic area, but in the earlier stage of thickening of the glia trabeculae, the myelin ring appears only thinned and atrophic and gradually lost. A low-power view of such an area gives the impression merely that the myelin sheath is faintly stained compared to the normal tissue, the transition to which is more abrupt than the glia-stained sections would indicate. In Marchi-stained preparations of the fully developed area there is a complete absence of any indication of myelin degeneration.

It must be noted, however, that in some areas which also give the impression of a primary glia change, which has gradually encroached upon the nerve fibres and led to the dissolution of the myelin, compound granular cells have been found at the periphery of the areas and in the walls of the small vessels within it, and also for a time very isolated examples in the tissue itself. Such cells are always very isolated and give the impression that the process has been a slow one, and that somehow, in the gradual withdrawal of these cells from the tissue spaces into the lymph channels, a few have been left behind.

Such areas, from the slowness of the demyelination process, the absence of all, or almost all, signs of reaction except in the neuroglia tissue, and the frequent absence of granular cells, may be looked upon as evolving gradually to a complete sclerosis without any intermediate stage of fat granule cell myelitis.

*B. Evolution of an Actual Sclerotic Area through a Stage of so-called
"Fat Granule Cell Myelitis."*

(a) Spinal Cord.

(i) Nerve fibres cut transversely (Figs. 8-12; 179-184).

It will be convenient again to take an area in the ventral third of the posterior columns of the cervical cord, for such are very numerous in our preparations. To trace the gradual changes by which the normal tissue is replaced by sclerotic tissue it will

be necessary to refer to stages. These, it must be admitted, are somewhat artificial, yet they are marked in general by definite histological characteristics.

(1) The first indications of change (Fig. 8) are best brought out by Van Gieson's stain, and seem to us related to the glia cells. The normal spider cells show a distinct enlargement not only of their nuclei, but of their protoplasm and protoplasmic processes, and the small darkly-stained glia nuclei in the tissue also show a lighter staining. As yet there is no proliferation of such cells, nor of any of the tissue cells, *e.g.*, cells in the blood-vessel walls. Very closely related to this glia cell enlargement is a change in the nerve fibres and in the glia reticulum. This seems to start in a slight oedematous swelling of the tissue meshes, a swelling and faint staining of the myelin sheath, and a diffuse pink staining and swelling of the contained axis cylinder. These alterations are very slight, and though analogous in kind to the similar changes found in acute myelitis, are not so in degree. The small capillaries are dilated and engorged with blood, and show a slight participation, in the dilatation of their adventitial lymphatic spaces, in the slight oedema of this localised area.

This swelling of the structural elements increases till large protoplasmic glia cells are formed, some of which show indications, in the presence of two nuclei, of a previous mitosis, though we have never been able to recognise definite mitotic figures, and it is possible, as many writers assert, that a direct division of such cells may occur. The myelin sheath of individual nerve fibres is so swollen and faintly staining as to be unrecognisable; around others only a faintly-staining ring of myelin can be found, and in some the swollen axis cylinder lies apparently free at one side of the distended glia meshes. Under low power the nuclear content of this area is already definitely increased. These nuclei belong in part to the enlarged spider cells, in part to the swollen glia nuclei, and in small part to an increase in the endothelial nuclei of the vessels, and, where a distinct adventitia is present, to an increase in the lining cells of the adventitial spaces. The share these vessel cells take is extremely difficult to decide, for numerous areas have been examined in which, under low power, the affected tissue was found distinctly to contain more nuclei, and yet none could be traced to any change in the cells of any of the vessel walls.

(2) The next steps in the process (Figs. 19 and 179) are characterised by the presence of a few large "epitheloid" cells, which, after the extraction of their contents with alcohol, appear vacuolated. This stage may be termed that of a commencing formation of fat granule cells. At first these are very isolated in the tissue, but as increasing numbers of myelin sheaths undergo degeneration their number increases very rapidly. The development of such cells at this stage must be largely traced to an increase in size of the small darkly-staining glia nuclei: the change in the nucleus of, and the increase in the protoplasm around, these cells may be followed till round cells are found, with a central nucleus in which the chromatin structure is quite visible and, with the protoplasm, taking a faint hæmatoxylin tinge. Such a cell is seen in Fig. 9, lying in the bay formed by two protoplasmic processes of an enlarged, multi-nucleated, spider cell. From such a cell all transitions can be traced to the fully-developed fat granule cell, in which the protoplasm between the vacuoles stains light purple, and its outer rim forms a distinct membrane. When this stage is reached, it is found that numerous glia spaces appear empty, or are occupied by fat granule cells in process of development; that the long ramifying processes of the enlarged glia cells extend for long distances and frequently envelop the fat granule cells; that numerous naked, swollen axis cylinders are found attached to the original glia meshes, which are now only faintly visible; that the nuclear increase is largely related to the presence of deeply-staining nuclei with a small amount of protoplasm; and that all the blood-vessels in this area are dilated and engorged with blood.

Slightly later in the development of the process, it is found that as the fat granule cells increase in number, there is often a definite reaction both in the endothelium of the small vessels and in the adventitial wall of the pre-capillary vessels. A special study was made of the recent areas to endeavour to trace proliferating endothelial cells in the vessel walls and their possible migration into the tissue. In many capillaries it seemed that the endothelial cells had proliferated and detached themselves from connection with the vessel. Some of the nuclei were perpendicular and oblique to the vessel wall, and appeared as if passing into the surrounding tissue. In the immediate neighbourhood of the vessels were frequently found, especially in cerebral

areas, small groups of cells, similar in their nuclear structure to the cells in the vessel walls. Stages could be traced in the further development of these into cells with distinct zone of protoplasm, which in its turn appeared vacuolated till true-fat granule cells were formed. It is thus seen that at this stage of maximum development of fat granule cells, the cells of the blood-vessel walls take a share in their formation, while at an earlier date they seem to arise from the proliferation of the small glia nuclei. Everywhere in the area were numerous small vessels, which give the impression of a new vessel-formation, but this might well be only an apparent increase, because all are so dilated and perceptible. In the cerebral areas this impression was much more marked than in the cord area, where the general architecture of the tissue seemed retained, in spite of the large increase in the cell elements.

(3) Following this is a stage (Figs. 10 and 180) in which the formation of fat granule cells has reached its maximum. The whole affected tissue seems permeated with these characteristic cells, which lie not only in every possible tissue space, but fill the adventitial lymph spaces of all the vessels within the area—even the smallest capillaries being surrounded by a uniform cellular ring, which gives the cross-section a very characteristic appearance. This stage may be termed that of a granular cell myelitis, and is the type of area we have described under heading 2 as representing the so-called acute multiple sclerosis. The presence of the fat granule cells in the vessel walls indicates that the cells containing the products of the disintegration of the myelin are already commencing to be removed in the lymph sheaths of the vessels. Probably as a result of the presence of these foreign bodies in the lymphatics there is a reaction in the cell elements of the adventitia, with the production of a certain number of small, round cells, with darkly-staining nuclei and a small amount of protoplasm. These cells can be recognised in the adventitia, together with the presence of the fat granule cells, and it is at this stage that the tissue gives the appearance of an inflammatory reaction in the vessel walls. Especially under low power, when the significance of this nuclear accumulation is not recognised, it seems that the tissue bears all the signs of an infiltrative myelitis. As the process advances, the protoplasm around the nuclei increases in amount, and the cell content of the adventitia can be differentiated into its various constituent elements (Figs. 264-266): (1) the

fat granule cell, with its vacuolated protoplasm and its nucleus, which has now undergone regressive changes and appears darker, and its chromatin texture denser, and later still the whole nucleus becomes crenated and fragmented; (2) the large vesicular nucleus of the proliferated endothelial cells; (3) the darker, smaller nuclei of the proliferated connective tissue elements of the adventitia; (4) small lymphocyte-like cells whose nucleus is scarcely to be distinguished from the former.

(4) The stage succeeding this (Figs. 10 and 180) may be termed that of a commencing fibril formation. Up till now the tissue at first glance appears as if large round nucleated elements had simply distended the glia meshes and taken the place of the nerve fibre; that these were specially numerous around the vessels; and that in place of the few spider cells of the normal tissue with the delicate glia reticulum, numerous large, proliferated, frequently multi-nucleated glia cells had arisen, whose long-branching processes entwined between and around the fat granule cells.

It is at this stage that there appear the first glia fibrils, as distinct from the glia cell protoplasmic processes. This fibril formation is beautifully brought out by Heidenhain's iron-hæmatoxylin stain, and specimens stained by this method and Van Gieson's method will be drawn upon for the description of the gradually increasing sclerosis of this area, which has so many of the characters of an area of acute myelitis. The essential feature of the rest of this evolution is the further development of the fibrillar glia tissue, which takes place at the expense of the protoplasm and protoplasmic processes of the large spider cells. The lateral margins and terminations of the processes are the first to develop into differentiated deeply-staining filaments, which at first retain their connection with the cell body, but as the differentiation is completed they become independent of the process and of the cell body, but retain a close relation to the cell nucleus. The nuclei seem finally to be the nodal points from which the fibres radiate. The evolution of these fibrils will be later more fully described, and here it is sufficient to state that these fibrils gradually assume a longitudinal position parallel to the longitudinal direction of the original nerve fibre. They are found gradually to increase in amount, and to interlace and almost to form a sheath with elongated meshes around the fat granule

cells. On cross-section of the nerve fibres it is difficult to trace this, but it can be seen that the fat granule cells are gradually compressed by the increasing fibril formation between them. It is probable that this compression aids the suction influence of the lymph flow in drawing the fat granule cells out of the tissue spaces into the lymphatic spaces. Many of the large protoplasmic glia cells are now found to have their protoplasm entirely transformed into fibrils. Along with the increase of the fibrils and the diminution of the fat granule cells is the tissue spaces, it is recognised that many of the axis cylinders which have survived the swelling have now returned to their former volume and are more readily recognised than at an earlier stage, lying in the meshes of the condensing tissue.

(5) The next stage, that of advancing sclerosis (Figs. 11, 12, and 181), is one which seems to occupy a long time. It is a gradual increase of the fibres, which seem able to become more abundant, possibly through fibrillation, even after their emancipation from the cell protoplasm. It is further a gradual diminution in size of the glia nuclei, and an increasing withdrawal of the fat granule cells from the tissue spaces, till only isolated examples are found. These may remain for a long time as vacuolated or granular spaces in the dense tissue, their nuclei very crenated or lost, and their membrane no longer recognisable. All the vessels, not only in the area, but the vessels radiating from it into the adjoining tissue, have their adventitial spaces crowded with similar cells. When the fibre formation has developed slowly and regularly, the pathological increase has taken place, according to Weigert and also in my experience, in a longitudinal direction, so that the transverse section represents the glia fibrils as fine granules, which surround the remaining preserved axis cylinders. Most of the glia cells have undergone regressive changes, but some large examples are still found.

(6) The final stage, that of complete sclerosis (Figs. 183-184), can scarcely be separated from the former: its complete evolution must be a very slow one, but the final result is a tissue which cannot be distinguished from that described under 1, as an old sclerotic area. In this area no fat granule cells remain either in the tissue spaces or in the vessel walls; the glia nuclei are mostly small, and may be fewer in number than in the normal cord; and the fine granular points, representing the fibrils, surround

the axis cylinders, giving the impression that a fine fibril formation has taken the place of the degenerated myelin sheath.

The blood-vessel changes, during the advancing sclerosis of the tissue, correspond to a very gradual condensation of the vessel walls (Figs. 272, 273). The fat granule cells are gradually drained away, leaving the adventitial spaces still dilated. Many of the nuclei of these cells are left, crenated or broken up, in the adventitial wall, and add to its nuclear abundance. The numerous nuclei at this time may be attributed to several sources, but the small round cells predominate. As the surrounding tissue becomes more and more dense, all the walls of the vessel seem to fuse and become homogeneous, and with Van Gieson's stain take a faint pink or yellow tinge. The outer layers of the adventitia remain for a long time separated, and contain cells of various kinds, granular debris, and blood pigment, but finally these all disappear, leaving a dense, homogeneous ring, in which almost no cell element can be recognised, except a very rare endothelial cell.

The glia changes correspond to the age of the process; on the one hand glia cell proliferation is found, and on the other glia fibril formation, and the tissue has altered to a dense felt-work. It is undoubtedly the large, pathological spider cells that produce the fibrils, and then in great part the nuclei perish. To such a disappearance and degeneration is to be traced the comparative nuclear poverty of the old sclerotic area.

It may be noted here that the glia fibril formation rarely takes place so uniformly parallel to the longitudinal direction of the nerve fibres. Where the large lateral blood-vessels course inwards from the surface at right angles to the long axis, they interrupt the direction of the fibrils. This may partly explain the almost constant radial arrangement of the glia fibrils around blood-vessels in an old sclerotic area. When, too, a greater degree of degeneration has occurred or a more rapid proliferation, the resulting glia fibrils will run much more irregularly (Fig. 189), and, especially round the blood-vessels, will form the tourbillons or whorls so frequently found in sclerosed posterior columns, and also in the lateral columns.

The above stages may be briefly described in the following terms, which characterise their dominant feature:—

(1) That of a commencing reaction of all the tissue components.

(2) That of a glia cell proliferation and a commencing fat granule cell formation.

(3) "Fat granule cell myelitis."

(4) That of a commencing glia fibril formation.

(5) That of an advancing sclerosis.

(6) That of a complete sclerosis.

In Weigert sections of an area in its process of evolution it is found that, in the earliest stage, the low power indicates little alteration, but a higher magnification shows that the myelin is swollen and diffusely and densely stained, and has not the clear, ring-like defined character of the normal myelin sheath. In the second stage this is even more marked amongst some nerve fibres, while others have their myelin sheath broken up into a group of globules, and still others show an almost complete absence of myelin within the area, but many of the fat granule cells contain granular globules that retain the hæmatoxylin stain, and still other granules and even fragments so stained are found free in the tissue (Fig. 238). In the succeeding stages the Weigert picture within the area is an entirely negative one.

Marchi-stained preparations show, even in the earliest stage, traces of a commencing degeneration of the myelin of the swollen nerve fibre, and frequently within this can be recognised, especially in sections counter-stained with safranin, a swollen axis cylinder. In the second stage the picture is very definitely one of degeneration in most of the affected nerve fibres, while the few fat granule cells present are filled with fine granules of a Marchi-staining substance, and numerous other cells show a commencing fat granule formation in their protoplasm. In the next stage the whole area is beset with large black formations—fat granule cells—scattered through the tissue and grouped around blood-vessels and septa (Fig. 143). In the two succeeding stages the Marchi reaction is confined to the remaining fat granule cells chiefly around the vessels (Fig. 145), while in the final stage the sclerosed tissue stains lighter than the normal, and the picture, like the Weigert one at this stage, is again a negative one.

Reference must be made to the transitional zone of such an area ere its complete evolution can be understood. The area which served as the illustration for our description was a

roughly triangular one, with its base to the posterior commissure (*cf.* Figs. 90 and 186). This well-marked layer of dense glia tissue around the central canal seemed to act as a barrier to the further development of the area in this direction, but at its point of greatest development the two lateral sides curved gradually to the apex about the middle of the posterior septum. Along these lateral sides there was a very gradual transition to the normal tissue which bordered the posterior horns. In this transition zone the stages were very similar to those described within the area, but were later in their development, so that, for example, when the fibril formation had commenced within the area, there was still nothing of it to be seen in the transition zone. It was, however, in the two final stages that the transition zone was most marked, for here, long after no trace of fat granule cells could be recognised even in Marchi preparations, there was a well-marked peripheral zone of such cells, and in this zone the vessels still retain traces of fat granule cells in their adventitia at a stage when the central sclerosis has been long complete. The presence of this zone containing fat granule cells and other changes characteristic of an earlier stage of development, *e.g.*, degenerating nerve fibres and proliferated glia cells, justifies the assumption that the process develops excentrically. When the transverse section of a sclerosed area in Marchi sections shows up lighter under low power than the surrounding normal tissue, and no degenerating fibres nor fat granule cells can be found either in the area itself or within the vessel walls at its extreme limit, the impression is given that the area has reached its climax of development, and that the process is stationary. All the products of degeneration have been removed and the glia fibril formation has also reached its climax, except in the transitional zone, which seems also stationary. Here a nuclear proliferation is very evident, mostly of small round cells; the nerve fibres have a thin, faintly-staining myelin sheath, but not a degenerated one; and it can be seen that the glia cell enlargement is continued into the normal myelinated tissue around—the glia cells showing an enlargement of their protoplasmic processes, without any apparent change in myelin sheath or axis cylinder.

(ii) Spinal cord area—nerve fibres cut longitudinally.

The series of drawings (Figs. 1-4) and photographs (Figs. 155-166 and 167-172) of a part of an area in the posterior columns cut

in longitudinal direction almost sufficiently indicate the nature of the changes. A glance at these figures shows the two outstanding features of the earlier process: the development (1) of an enormous number of large glia cells with a wide zone of protoplasm and numerous branching processes which quite mask the outline of the cell body, and (2) of rows of fat granule cells which tend to occupy the position of the degenerated myelin tubes. The latter figures show (1) the gradually increasing fibril development at the expense of the glia cell protoplasm and processes, (2) the increasing diminution of the fat granule cells in proportion to the increase in fibrils, and (3) the presence of large numbers of axis cylinders in this fibrillar tissue.

Here also the first change visible is an enlargement of the protoplasmic processes and nucleus of individual spider cells. This is closely followed by a swelling both of myelin sheath and axis cylinder, both of which swell out to occupy the whole space of the distended glia meshes, without, however, breaking through the glia reticulum which forms the meshes. Together with this change in axis cylinder and myelin sheath is an increase in the number of the normally present small glia nuclei, which may be seen, often in short rows, lying between the swollen nerve fibres (Fig. 155). It is in such a section that it is possible more easily to follow the gradual increase in size of these cells and their development into the first fat granule cells, which lie closely applied to the swollen and degenerating myelin sheath. At this stage numerous dilated longitudinal capillaries and larger transverse and oblique vessels can be recognised in the affected tissue.

The development of the large spider glia cells occurs *pari passu* with the development of the small glia nuclei, and long rows of hypertrophic cells (Fig. 209) of very varying shape may now be found, their long-branching processes passing in between the degenerating nerve fibres. The increase in the formation of fat granule cells takes place in a very close relation to the number of the degenerating fibres, and soon the whole tissue is composed of these rows of the two cell elements, pushed aside amongst which may be found very swollen axis cylinders or granular remains of such (Figs. 1, 156, and 168).

The stage of "fat granule cell myelitis" has now been reached, and the description of the early area described under heading 2 applies to this area. The further evolution to the old sclerotic area

described under heading 1 consists in the further development of the fibrillar glia tissue already described in transverse section. Here, however, it is much more easily recognised how the fibril formation tends to take place in a longitudinal direction. When the tubular rows of fat granular cells are fully formed, the glia cell processes twine around them and, when the fibril formation commences, the fibrils not only form between the rows, but pass in transversely or obliquely between groups of three, four, or more granule cells, thus forming interlacing bundles of fibrils (Figs. 2 and 159). In the elongated meshes formed by this interlacing there would seem to be a gradual compression of the granule cells, for many seem to undergo a gradual breaking up *in situ*. The nuclei become hyperchromatic and crenated, and the definition of the cell membrane is lost, till finally only nuclei are left with a few traces of vacuolated protoplasm around them. These nuclei remain long in these glia meshes, and add to the nuclear abundance of the areas at this time. With the gradual disappearance of the fat granule cells the interlacing bundles of fibrils gradually become more parallel (Fig. 160); the glia cell nuclei diminish in volume as their protoplasm is differentiated into fibres; the axis cylinders, which have survived the early swelling, are now more defined (Fig. 161), though many are still swollen and weakly stained; and, finally, the numerous vessels in the fibrillar tissue, which up till now have had their sheaths widened and filled with cell elements, gradually undergo the regressive changes already described.

For the stage of complete sclerosis, reference may be made to the area described under heading 1 (Figs. 3, 4, and 161-166).

Weigert sections of such an area, during its evolution, show the successive changes of diffuse swelling of the myelin sheath, gradual loss of staining, and, finally, a negative picture with at first numerous fragments and granules, retaining the hæmatoxylin stain, scattered throughout the tissue. The transition zone is often a broad one on all sides, and both at its upper and lower limits it is wedge-shaped, the lateral margins of the wedge forming a zone in which all the changes are characteristic of an advancing process. If such an area undergoes complete sclerosis, this wedge-shaped zone shows (1) a looser structure than the central area—its fibril formation being not nearly so dense—and (2) a marked nuclear proliferation, of small and large glia cells, which extends for a short distance into normal tissue.

Marchi sections (Figs. 149-153) and frozen sections, stained with Scharlach R. (Figs. 18-20), reveal very beautifully the gradual changes in a degenerating nerve fibre — the presence of fine granules and globules or small and large irregular particles. Those changes may take place in a portion of a nerve fibre which in its subsequent course stains normally and then again shows degenerative changes. At the stage of maximum development of fat granule cells both Marchi and Scharlach R. specimens show their almost tubular arrangement in the tissue, and as the tissue becomes cleared, the arrangement in long rows around the longitudinal vessels, and in the vessels, cut transversely, which reach the pia. In the latter their numbers gradually diminish as the pia is reached, and those that are left pass into the lymph spaces in the inner layers of the pia (Fig. 145). In the stages of advancing and complete sclerosis, fat granule cells are left for a long time in the walls of the vessels radiating from the sclerosed area: the transition zone also shows a complete layer of such cells in the tissue spaces. As the process becomes stationary, these also are drained away, leaving the whole area and the transition zone staining lighter than the normal tissue.

(b) *Cerebral area: e.g., in the central white matter.*

(i) Nerve fibres cut in various directions (Figs. 5, 6, and 191-199).

It is when we come to trace the evolution of such an area that we recognise how complicated is the resultant sclerosis. The nerve fibres, instead of running parallel in one direction, here run in every possible direction—not only in straight lines, but in curves. It is a web in which, in addition, the threads in bundles and groups of bundles cross each other and also curve round to cut across the longitudinal, transverse, and obliquely-running threads. Those who have studied, by means of the various elective stains, the structure of an area, *e.g.,* of the central white matter, have realised how difficult it is to define the plexus formed even when only one of the structural elements is stained. When, however, in consequence of the degeneration of the myelin sheath, or of both myelin sheath and axis cylinder, their place is taken by a dense fibrillar tissue, which more or less follows the varying direction of the lost fibres, it will be seen how aptly such

a tissue may be described as an inextricable tangle, in which the finest meshes are invisible under low power.

Here also the first stage is one of reaction of all the tissue elements: at times it seems as if the glia cell enlargements were distinctly the primary change; at other times it is equally clear that a myelin sheath alteration, or even its complete dissolution, has preceded the glia cell change; and again it occurred that both changes are coincident. Heidenhain's iron-haematoxylin stain is of great service in allowing both the change in the myelin sheath and the change in the cell elements to be recognised in the same section, and frozen sections were found likewise of great help in coming to a decision as to which was the primary tissue element involved. It was distinctly evident that both changes preceded any recognisable alteration in the vessel walls. The frequent centrally-placed vessel and all the capillaries and pre-capillary vessels were dilated and engorged. In Van Gieson's stain the smallest capillaries are brought out distinctly, for the outer membrane of the endothelial cells stains intensely with fuchsin—and, in addition, a fine double contour, frequently present, gives the impression of a distinct adventitia or elastic coat even to these fine capillaries. At this stage, also, there is an increase in the small normal glia nuclei of the cerebral white matter, and an indication of a commencing enlargement of the nucleus.

At a later stage the large glia cell proliferation (Figs. 13 and 264-266) is the most outstanding characteristic: the rapid appearance in such large numbers of those large protoplasmic cells with homogeneous protoplasm and long-branching processes is closely related to the numerous dilated vessels everywhere present in the affected tissue. It would seem that it is the glia cells, which normally lie within and close to the peri-vascular glious sheath, which first enlarge and proliferate, and several of their broad processes end in the outer wall of these vessels. Closely following this proliferation both of the large and small glia nuclei, appear the first fat granule cells, which in size and structure closely resemble those found in the cord area (Figs. 13, 191-193). These likewise develop in such profusion that soon the whole area is permeated by them, and under low power the tissue appears fenestrated and areolar—the spaces being occupied by round nucleated elements, and the intercellular spaces being filled by large protoplasmic glia elements whose branching processes

again entwine around the cells. When the climax of fat granule cell development has been reached, Weigert and iron-haematoxylin sections indicate the almost complete absence of myelin within these areas; the progressive changes in the nerve fibres are not so easily followed as when the nerve fibres run only in one direction, but both Weigert and Marchi sections show the characteristic appearances of an early degeneration, the Weigert sections giving a myelin sheath diffusely or faintly stained, and the Marchi, the different stages of its disintegration. At the stage of "fat granule cell myelitis" the whole area is again beset with granular cells, both in the tissues and in the vessel walls. Marchi preparations (Figs. 68, 131) show the structure and arrangement of these best, but cell stains, and Marchi sections counter-stained with safranin, in which the fat has dissolved out of the fat granule cells—leaving the skeleton structure of the tissue—show best the relation to the other tissue elements.

With the onset of the fibril formation, the walls of the blood-vessels, both within the area and leading from it, are even more densely packed with fat granule cells, amongst which may be found other nucleated elements, the result of the reaction of the adventitia to the cells in its lymph spaces. It must again be emphasised that it is areas at such a stage of development that have given the justification for regarding the process as developing on the basis of an acute myelitis, for the cell infiltration of the vessel walls and the presence of fat granule cells in such abundance in the tissue gives all the appearance of a softened and infiltrated area. At an earlier stage we have seen that the blood-vessel changes are not constant, and that only on the onset of the removal of the fat granule cells in the lymphatics is there any definite cell infiltration of the vessel wall. Later, as we shall see, the blood-vessel changes again recede in their prominence. Figs. 192 and 195 give a clear idea of the commencing fibril formation and the gradual separation of the fat granule cells by the fibrils, and Figs. 266 and 267 give a conception of the vessel changes at this stage.

The stage of advancing sclerosis is well represented by Figs. 6, 193, and 196. It will be seen in the low-power view that in a large area the sclerosis advances by no means uniformly—small areas being present in which there is an almost dense tissue, while others still contain numerous fat granule cells. The blood-vessel

changes correspond very closely to those described in the spinal cord area, and will be more fully referred to in a later section. Weigert sections give a completely negative picture within the area; Marchi sections give a negative picture in the sclerosed parts, but the fat granule cells are well brought out in the tissue spaces and surrounding the blood-vessels. The further development of the fibrillar tissue, at the expense of the glia cell protoplasm, and the gradual removal of the remaining fat granule cells, leads to the formation of a dense sclerosis, the meshes of which are so fine that under low power almost no spaces can be recognised (Fig. 194). Within this area the glia nuclei gradually diminish in size and number, and, finally, they are fewer than in the normal tissue of the neighbourhood. Both Weigert and Marchi sections give a completely negative picture of such an area—the areas standing out clear against a dark background (Figs. 122-124, 227). Glia stains, on the other hand, give a dense positive picture—the area standing out deeply stained against a lighter-stained background (Figs. 228 and 231).

The transition zone of such areas must now be referred to. Here, also, we have a gradual but slower evolution of the same changes as within the area, but the sclerosis never reaches the stage of a final, dense meshwork. In this transition zone are found at successive stages degenerating myelin fibres, with hyperplastic glia cells and fat granule cells both in the tissue and in the vessel walls—signs of an advancing process, which frequently remain long after the sclerosis within the area is complete. When, finally, all indications of myelin degeneration have been removed, the transition zone is markedly different from the adjoining sclerotic tissue on the one side, and the normal tissue on the other—from the former in the lesser degree of glia fibril formation, and from the latter in the diminished number of myelin fibres, and the thinness of the ring of myelin around such as persist in this zone. Again, as in the cord area, there is a marked small glia cell proliferation in this transition zone (Fig. 233), together with a few enlarged glia cells (Fig. 234). The latter may also be found extending for a short distance into the surrounding, otherwise healthy tissue. In this description of the evolution, few references have been made to the persistence of the axis cylinders. Nowhere is the distinction more difficult to draw between glia fibrils and axis cylinders than in such cerebral areas—not only with the ordinary

diffuse stains, but with Bielschowsky's method, which here so frequently stains the glia fibrils—a finding which numerous writers have noted in cerebral areas. In two areas stained by Cajal's method, there were found a very large number of persistent axis cylinders; in one case (Fig. 260) the whole fine reticulum of axis cylinders and their branches were retained, but in three other small areas no axis cylinders could be found. By the diffuse stains, in numerous instances, it could be asserted that almost the whole axis cylinder content of the area was retained.

- (ii) Cerebral area cut in longitudinal direction of the nerve fibres (Figs. 200-207).

Such an area shows no essential changes from those described in the longitudinal direction of a spinal cord area. In my experience the preserved axis cylinders were very much less numerous than in the cord. Fig. 252, taken from an area at the base of a medullary ray, shows the appearance presented in a specimen stained by the Bielschowsky method.

4. *Other Types of Areas.*

So far the histological description has been confined to areas which may be looked upon as typical of a late and of a recent process respectively, and to the evolution of a sclerotic area, on the one hand by a gradually increasing hyperplasia of the glia, and on the other by stages which include the formation of a large number of fat granule cells. As different stages occur in the same case, and as the process by no means always follows the same uniform course of development, it will be evident that the histological picture met with is a very varied one. Yet before a comprehensive view of the complete process can be obtained, it is necessary to add to this complicated picture a brief description of other areas—areas so frequently met with as to constitute types almost as definite as those already taken of an old and a recent process.

(a) *Areolar Areas.*

The "areolierte" areas of German writers. At first sight it would be natural to relate such areas simply to an arrest of the fibril formation before a dense sclerosis had taken place. An examination of the transition zone around most of the old areas,

where the process has become stationary, and yet the fibril formation is not so abundant as in the centre of the area, would seem to justify this explanation. A close examination, however, shows that this cannot be accepted as the constitution of all such areolar tissue. Under a high magnification the glia is found to be often relatively unaltered, and to show only a distension of the network. Wide meshes are thus formed, with cells at the nodal points of the network, and frequently larger spaces result from the opening into each other of adjoining meshes by the breaking down of the glia fibres separating them. Within the meshes the myelin sheath has disappeared, leaving the axis cylinder at one margin, or this also has disappeared, and the space is empty, or contains only granular remains of regressively changed fat granule cells. An area of dense glial sclerosis is frequently quite completely surrounded by such a peripheral areolar zone, which interposes between it and the healthy tissue. This peripheral zone contains numerous nuclei and dilated vessels, with nuclear accumulations in their dilated adventitial sheaths: its mode of formation is to be distinguished from that of the transition zone already described—the latter being distinctly an arrest of the development of the fibrils before complete sclerosis had occurred, while this areolar zone around a compact area, and the areolar areas described in this paragraph, are to be traced in all probability simply to an acute and rapid degeneration of the nerve elements, with a distension of the original glia meshes before fibril formation had occurred. In the spinal cord such a distension of the glia meshes, swelling of the myelin, and distension of the adventitial lymph spaces of the vessels extends sometimes over the whole transverse section of the cord. At other times at the periphery of the cord we get a widening of the glia meshes. This is only in small part due to the retraction under the influence of hardening agents. The abnormal dimension of the interstices points to the presence of liquid in excess in the tissue. A primary degeneration of the nerve elements, at least of the myelin sheath, as an explanation of “areolar” areas has been assumed by those who see in such areas a proof of the change in the myelin sheath as the essential and primary feature of disseminated sclerosis. It is not denied that later, if there is time, a proliferation of the glia may be associated. On the other hand, Redlich and others see in “areolar” areas a process quite distinct from the essential substratum of a typical sclerotic area.

It must here be added that very large numbers of the areas found in all the cases, especially cerebral areas in which the process was quite stationary, as far as the degeneration of the myelin at the periphery was an indication, showed under low power a much less dense sclerosis than that described as compact sclerosis. In such areas fat granule cells were absent both from the tissue spaces and the vessel walls; Marchi and Weigert sections also gave a completely negative picture, but cell stains and glia stains showed that numerous large, multi-nucleated glia cells were still present, and that each was the central point from which a marked fibril formation radiated. This fibril formation, however, had not been sufficient to form the inextricable tangle of the denser areas, and yet was quite distinct from the simple distension of original glia meshes of the areolar areas (Figs. 197, 198). We thus draw a sharp distinction between these two forms, which have both received the name of "areolar" areas, and reserve this name for the areas in which the original glia meshes are retained or distended, and reserve "areolar zone" for the zone sometimes found around compact areas, which has a similar structure.

(b) *Peri-vascular Sieve-like Areas.*

Non-myelinated areas are frequently found, especially in the brain, in which large open spaces are met with around all vessels found within the affected area. The wall of this space is, on the side of the nerve tissue, represented by a dense ring of glia fibrils, often with very few nuclei. The walls of these vessels seem often little modified: they may be slightly thickened and infiltrated, but the essential change is a separation of the constituent elements of their adventitial sheath so that the connective tissue fibrils stretch across and form a very wide-meshed reticulum between muscle coat and condensed glia (Figs. 278-280). Within these meshes a fine coagulum (Fig. 271) is frequently found, together with a few cells, often containing pigment or the remains of fat granule cells. The vessels thus affected lie together in groups, and give the tissue a sieve-like character, which, when advanced, has been termed "l'état criblé." The impression is received that it is the branches of one vessel stem (Fig. 278) that are concerned in this change. Around each vessel the myelin fibres are dissolved, the glia meshes are widened, and both large and small glia cells are proliferated (Fig. 279), and the areas affected, around each vessel,

usually coalesce to form a more or less large irregular non-myelinated area. Frequently half the vessels affected lie within non-myelinated tissue, and half within tissue in which the myelin as yet stains normally (Fig. 280). Borst considered that these areas are the result of a circumscribed peri-vascular lymph congestion, and that they are a pre-stage of areas of true dense sclerosis, which, he considers, develops on the basis of a lymph stasis in the tissue.

(c) "*Markschattenherde*."

As a rule, at least at the commencement of the process, sclerotic areas have a very limited longitudinal and transverse extent. This characteristic has given to the disease the name "insular sclerosis," because the areas appear isolated. We have seen, however, that areas frequently coalesce on one or several sides, and the original outlines are quite indistinct. In addition to this, there are often found between individual non-myelinated areas, extensive patches which, with Weigert staining, show a weak staining of the myelin. The fibre bundles are distinctly perceptible and yet scarcely stained: this is well brought out on longitudinal sections, where it is seen that the fibre layers correspond in arrangement and position to the normal bundles, and one can recognise the immediate transition of normal fibres into those with deficient staining. Such areas, in which the myelin sheaths are simply shadows or very thin, adjoin areas of complete demyelination, and may involve the whole transverse section or the longitudinal section over several segments. Alzheimer looks upon this deficient staining as a condition of the myelin sheath antecedent to degeneration. Marchi staining sometimes gives a very extensive early degenerative process, and there may be an equally extensive commencing glia proliferation. Yet this change may sometimes indicate a simple atrophy which results in a progressive reduction of the volume of the sheath without affecting the remaining myelin. The process might then be looked upon as a slumbering one, and not necessarily immediately antecedent to degeneration. In the former case the glia proliferation would be more abundant, as the slow, chronic stimulus would be more likely to lead to a greater reaction in the interstitial tissue. Thus Volsch has described areas in which there was a very extensive "*Aufhellung*" of the myelin sheath, which was associated with an equal or even greater degree of glia hyper-

plasia—a condition which he has termed “diffuse multilocular sclerosis.”

It may finally be noted that in Marchi sections there was frequently found, extending outside the sclerosed area uniformly over the whole transverse section of the cord, an early myelin sheath degeneration. This could be noted even in the nerve roots, and must, probably, be traced to the septic fever from which the patients suffered.

(3) HISTOLOGICAL CHARACTERISTICS IN SPECIAL SITUATIONS.

1. *White Matter.*

This has already been fully considered, as the areas described in the previous sections were all in the white matter of the brain and cord.

2. *Grey Matter.*

(a) *Central Grey Matter.*

Where the sclerotic process affects the grey matter of the spinal cord and the corresponding nuclei of grey matter in the medulla oblongata and pons, we find that the changes in their evolution correspond very closely to those in white matter in which the nerve fibres form a reticulum.

The myelin network quickly disappears, and in its place we get a thickened glia reticulum with large meshes and numerous hypertrophied glia cells. These spider cells, with relatively numerous processes, varying in length and thickness, give rise to a fibril formation analogous to that already described—the resulting meshwork being very close. The formation of fat granule cells is always much less marked than in the white matter; their size is smaller and the structure of their granules more delicate. This corresponds to the lessened quantity of myelinated fibres in the grey matter, but the process of their formation, the absorption of the degenerated myelin, their presence in the glia meshes, and their gradual removal in the lymphatic spaces of the vessels is quite similar to that elsewhere. The vessels passing from the borders of the grey matter into the white matter form often radial lines; the commissural vessels and the vessels in the anterior median fissure also take their share in the removal of degenerated products.

The ganglion cells in this reticulum seem to remain long preserved (Fig 247), and in this investigation changes, which could be distinctly traced to the sclerotic process, were found only when an advanced degree of sclerosis had been reached (Fig. 249). The cells undoubtedly remain long with their normal form and minute structure preserved. The processes first lose their structure and, as the sclerosis becomes denser, the cell bodies share in this change. In the later stages they show all the possible changes which are traceable to a slow simple atrophy from loss of function (Fig. 239) or from compression on the part of the developing cells and fibrils of the interstitial tissue (Fig. 249). Diffusely extensive changes in the ganglion cells (Fig. 241) should probably be referred to somatic general disturbances which accompany the disease, *e.g.*, the fever, anæmia, exhaustion, or direct septic absorption from bed-sores or a pyelitis, and can thus not be looked upon as specific to disseminated sclerosis. In numerous affected segments which microscopically showed complete demyelination, many of the ganglion cells showed no changes—completely normal cells being found alongside those with chromatolysis or atrophy.

As the glia reticulum becomes denser, and encroaches more and more on the ganglion cell processes and cell body, there takes place a gradual atrophy of the cell (Fig. 245). For a long time an atrophic, rounded, or oval cell, with no processes and no chromatophile granules, and usually without nucleus, may be recognised (Fig. 248). But gradually this faintly or diffusely staining body can no longer be recognised as a cell, and this dense tissue consists of the deeply-staining glia nuclei and the abundant delicate fibrils (Fig. 249).

It is frequently stated that sclerotic areas are never found solely in the grey matter, but that these are always extensions from sclerosis of the white columns. In serial sections, however, it could be proved that small sclerotic areas may be confined, throughout their whole longitudinal extent, to the grey matter. An affection of the myelin reticulum around and between one or other group of cells has frequently been the sole trace of demyelination in many sections of the lumbar cord. A peri-central sclerosis, limited at first entirely to the commissures, is often the starting-point of an extension into each anterior and lateral horn. The spread may take place forwards along each lateral margin of the anterior fissure or posteriorly along the posterior median septum

as a central line, or the extension may be along all of these planes, giving rise to a cruciform area of sclerosis, which in its further extension may involve the whole transverse section, leaving sometimes four lateral and symmetrical peripheral areas situated near the posterior and anterior roots respectively.

Undoubtedly, however, the affection of the anterior horn of grey matter of the cord starts very frequently from the extension inwards of an area of sclerosis situated at the margin between white and grey matter (Fig. 71). In the transition of an area from grey to white there is such marked glia proliferation that the transition can no longer be recognised. Müller looks upon this border line as an area in which the glia is more fully developed: this is certainly so in the region of the *formatio reticularis*, which is the most frequent site of development of a gradually increasing glia hyperplasia. The development of areas at this transition zone has been explained by other writers with reference to the breaking up of both central and peripheral vessels, and it is not difficult to trace the direct passage of a lateral vessel to such an area (Figs. 71 and 83), or of thickened commissural branches passing to areas in the lateral and anterior grey matter.

The definition of the area in the grey matter was never a sharp one: the nerve fibres of the reticulum passing into the area for very varying distances (Fig. 242). It was also found that where an areolar zone surrounded an area which involved both white and grey matter, it ceased almost abruptly at the transition and was never present in the grey matter.

When sclerosis affects the cranial nuclei, as is so frequently the case, the process may again be confined to the grey matter, but this is rare, and it is much more often an extension of a process which involves the floor of the IVth ventricle. The histological characters in no way differ from that just described. Fig. 247 shows an involvement of the hypoglossal nucleus.

Sclerosis of the olivary bodies and of the dentate nuclei leads to a gradual thinning of the lamellæ. In numerous specimens there is present a very advanced degree of sclerosis of the fibres entering at the hilum and passing to the grey matter before there is any noticeable involvement of the glia reticulum of the lamellæ, unless these are involved in a general extension inwards from the surface of the medulla or ventricle. The ganglion cells (Fig. 205), as far as it was possible to interpret their changes, seemed to under-

go a gradual and uniform structureless appearance, and to retain a deeply-staining nucleus till the cell body was almost completely atrophied.

Numerous areas at all stages of development were found in the basal ganglia (Figs. 95-97). In the evolution of these areas there is little distinctive to be added. The numerous bundles of parallel nerve fibres which pass through these ganglia and intersect each other frequently give to the resulting glia reticulum a more uniform appearance over short stretches. It was also noted that in such areas it was possible to prove how the process advanced without any relation to any conducting tract of fibres, and simply involved the immediately adjoining tissue bundles in whatever direction they ran. It was found impossible to relate any specific changes in the ganglion cells to the sclerotic process: these underwent a gradual atrophy as the fibril formation became closer. Very large and very numerous spider cells were found in all the early areas, and the fibril development was a very close-meshed one, the sclerosed tissue being formed by a web of very closely arranged fibrils oriented in all directions. The special changes in the branches of the lenticulo-optic and lenticulo-striate vessels will be considered later.

(b) Cortical Grey Matter.

It is now well recognised that the cortex is affected in disseminated sclerosis. Before the introduction of the Weigert medullated sheath stain, such areas were often overlooked, and by many their existence was denied. Two deductions may be drawn from this circumstance: the one, that cortical areas are difficult to recognise except with a medullated sheath stain; the other, the complement of the first, that the glia, axis cylinder, and cell changes in the cortical areas are very slight. Both deductions are, in my experience, justified. A very large number of portions of tissue from the cerebral convulsions in which macroscopically—after fixation in formalin or after mordanting in bichromate—cortical areas could be distinctly made out, were taken through both for celloidin, paraffin, and frozen sections. In only a small proportion of these could the demyelinated area be recognised in cell stains, even after the most careful comparison with the control Weigert or iron-hæmatoxylin-stained section. The cortical portion, which showed a complete absence of myelin (Figs. 128, 215), seemed

to show no other change related to the process of disseminated sclerosis. In comparison with the adjoining cortex on either side, with the cortex of adjoining convolutions, or with the cortex of an altogether different portion of the hemisphere, there seemed to be no change in the ganglion cells, glia cells, or axis cylinders. Yet there were many areas in which recognisable alterations in these structural elements in the demyelinated area did occur (Figs. 129, 216-226), and it is these which will now be described. It has seemed impossible to reach any explanation of why certain areas showed changes and others did not do so.

Minute areas were found in all the layers of the cortex, but before referring to these it will be simpler to trace the changes in an area which passes over from the subcortical white matter into the cortical layers. The earlier writers, as we have seen, asserted that this transition never occurred, and that the grey matter formed a barrier to the extension of the process. Sections stained with Weigert's glia method were largely responsible for this statement, for such preparations showed an almost abrupt cessation of the glia fibril formation at this border, and recent writers emphasise the complete absence of a glial sclerosis as one of the essential characteristics of a cortical area. Is, then, the process that attacks the cortex different in its nature and origin from that which affects the rest of the central nervous system? Those who see in disseminated sclerosis a primary proliferation of the glia must admit that in the cortex, at all events, this is not the origin of the process, and that a primary degeneration of the myelin sheath is often the sole change. The examination, however, of a very large number of sections from very numerous areas in several cases, by means of the Heidenhain iron-haematoxylin method, both in celloidin and paraffin and frozen sections, and a comparison of such sections from the same block of tissue, stained by Ford-Robertson's methyl-violet method, Scharlach R., Bielschowsky's silver impregnation method, and the routine diffuse stains, has led to the conclusion that, while a fibril formation in the layers of the cortex is proportionate in its development to the normal glia fibril content of the layers, a change in the glia cells and fine glia reticulum of the cortex is very closely related to the loss of its myelin fibre content.

In the area represented in Fig. 116 it is seen that the absence of the myelin affects a portion of the medullary white matter, and

the radiating fibres in the cortex. The demyelinated tissue is roughly wedge-shaped, with its broad apex in the white matter and its base on the surface of the convolution. Its outline is clearly defined from the surrounding radiating fibres. The great majority of areas in which changes in the cortical layers were found in association with subcortical changes were in the condition of recent areas. The structure of that portion in the white matter, therefore, was similar to that described under heading 2. The enormous spider cell proliferation and fat granule cell formation attained, at this transition border (Fig. 219), its maximum intensity, and passed over into the deepest layers of the cortex so that the border could no longer be recognised. Specially prominent was the development of the glia cell processes which attached themselves to the walls of the capillaries and pre-capillary vessels (Fig. 266). Each of these was surrounded by rings or layers of fat granule cells, and to the outermost of these the glia cell processes formed almost a radial arrangement. Each ganglion cell in the stellate layer and in the layer of the deep pyramids was in a condition of marked degeneration and atrophy. Many were mere ghost-like forms with no structure; in others only the large nucleus remained, and still others were replaced by nests of small round cells from five to ten in number (Fig. 211). The whole tissue of these deeper layers was so crowded with the two proliferated cell elements, large glia cells, and fat granule cells, and with the numerous dilated small vessels, that the ganglion cells were almost lost sight of. Where the area involved (Fig. 217) the Betz cells, these also were found to show all stages of degenerative change, and many of them had disappeared. The changes gradually receded in intensity as the upper limit of the deep pyramids (Figs. 222, 225) was reached: the cells in the granular layer were found to be surrounded by nests of small round cells (Fig. 223), but here the glia spider cell and fat granule cell formation was very limited. The fat granule cells present had also a finer structure than that in the deeper layers (Fig. 226).

The intensity of the process gradually lessened as the upper layers of the cortex were reached. Amongst the large pyramids were found still a large number of proliferated glia cells, but now the processes of these were of a fine, almost uniform calibre, and reached a very long distance from the small nucleus from which

they radiated. Marchi sections showed a very characteristic appearance in these layers. All the satellite cells, which are normally round and have no visible protoplasm, were found to have developed a protoplasm cell body, of various shapes, often spindle-shaped and star-shaped, and to surround with their processes the ganglion cell (Fig. 226). Their protoplasm was studded with the minutest black granules, and transition forms could be traced between those surrounding the ganglion cell and rounder forms lying free in the tissue spaces or in the sheaths of the larger vessels. The finest capillaries were surrounded by similar granular cells of varying shapes, which had evidently arisen from the rows of small round cells which in these cortical layers normally surround the capillaries. It is to these cells and to the very similar round cells in the white matter that Ford-Robertson has given the name mesoglia cells, and to which he attributes phagocytic properties. Of their phagocytic character, their protoplasm, loaded with fine fat granules, is a direct proof. Here also the fine uniform processes of the glia cells have a very intimate relationship to the vessels, especially to the very abundant capillary plexus in this region. When we reach the surface layer we find again a very abundant development of glia cells, many of which are multi-nucleated and in the process of fibril formation. Diffuse stains—for here specific axis cylinder stains failed—showed that numerous axis cylinders could be recognised, but that very many had perished in the dense zone of reaction.

To sum up briefly the changes in a recent combined subcortical and cortical area, it is sufficient to emphasise these related to the glia cell and fat granule cell development, for the myelin sheath and axis cylinder changes do not differ from those in other areas. In the transition from white matter to cortex the deepest layer of the cortex is no longer recognisable (Figs. 218-220). In the layer of the deep pyramids (Fig. 222) and Betz cells (Fig. 217) both glia cell proliferation and fat granule cell formation are still very marked: from the granular layer upwards, however, the intensity of the cell reaction is much less. Nests of small round cells are found around nuclear remains of atrophied ganglion cells (Fig. 233) and around others, and around the capillaries is found a fine fat granule formation in the satellite cells (Fig. 226), together with a very delicate glia fibril formation, which requires

the highest magnification to recognise. In the marginal zone there is again a marked glia cell and fibril reaction.

If the evolution of such an area be followed, it is found that in the white matter and deepest layers the process often follows the lines one would expect from the presence of the large protoplasmic potential fibril-forming glia cells. This sclerosis extends to involve the deepest layer of the cortex. Above this, in the layer of deep pyramids and Betz cells, the glia cell nuclei are left as the nodal points from which radiate a glia fibril formation which is insufficient to cause sclerosis; the fibrils, however, merge into a very delicate reticulum, which may be the syncytium claimed by Held to form the groundwork of the cortex. In the superficial layers of the cortex the long, uniform, delicate processes of the glia cells also unite with this reticulum, and in the surface layer the proliferated glia cells form fibrils which extend downwards—also to merge with the syncytial reticulum.

The ganglion cell changes in relation to these areas will be taken up under the heading of ganglion cells.

When an area is confined to the cortex, the changes are, as a rule, not nearly so marked, especially those in the deepest layers. The demyelination may reach from the surface of the convolution to varying depths (*cf.* Figs. 99, 103) even to the border between cortex and white matter. It may lie wholly within the cortex and cut through a portion of the radiation, or simply affect Baillarger's stripe. These areas are often in association with a markedly dilated vessel which penetrates from the surface almost to Baillarger's stripe, and a number of dilated changed smaller vessels lie within the area. Marchi sections of such areas show that the myelin sheath is not attacked as a whole: the black staining gradually increases to involve the whole myelin ring, and the axis cylinder shows by its swelling a distinct participation in the process. The ganglion cells in such areas show everywhere nests of glia cells around them: these changes are by no means confined to the actual demyelinated area, but are more marked there than elsewhere, and most constant in the granular cell layer (Fig. 223). The glia cells in the layer of the deep pyramids show the changes represented in Figs. 222, 225—a marked proliferation and fibril formation, which, however, is again insufficient to cause sclerosis. In the layer of stellate cells (Figs. 221, 224) also there are the same changes with a more marked

disappearance of the ganglion cell bodies, leaving only nuclei surrounded here and there by nests of cells.

3. *Peri-ventricular Sclerosis.*

This special localisation, noted by Charcot, Borst, Strähuber, Westphal, and others, has been emphasised as the dominant feature in the cases reported by Lhermitte and Guccione, Merle and Pastine, and also by Schob. It is of special interest in relation to the pathogenesis of disseminated sclerosis, and at once raises the question whether the development of the peri-ependymal areas may not be conditioned by the presence of the causal agent in the cerebro-spinal fluid itself. In horizontal sections through the cerebral hemispheres, there was frequently found a sclerosis which, macroscopically, seemed limited to the walls of the ventricles and very numerous sections at various levels were studied to determine the exact limits of this alteration, in what it consisted, and whether the apparently isolated areas in the adjoining grey nuclei or white matter were really offshoots from the areas on the ventricular surface. In all the cases which showed a peri-ventricular sclerosis, this was much more marked around the horns, especially the posterior horn (Figs. 23, 24), but sections cut at lower levels, *e.g.*, through the temporo-sphenoidal lobe (Fig. 29), horizontally or sagittally, showed the almost equally marked involvement of the descending horns on both sides, and sections of the hemispheres cut at levels immediately above the corpus callosum showed large round isolated areas in the central white matter, some of which, in serial section, proved their connection with the sclerosis of the roof of the lateral ventricle (Figs. 25, 26). It is thus seen that not only were the walls of the ventricle involved, to a varying degree, throughout their whole surface—lateral walls, horns, floor, and roof—but that this sclerosis extended inwards from the ventricular surfaces, forming a zone from one half a centimetre to 1 centimetre broad in the adjoining grey nuclei or white matter. Further, that from this zone at numerous points processes—sometimes finger-like, sometimes cup-shaped—passed deeper into the surrounding tissue. Such areas appeared naturally, in some sections, isolated from the peri-ventricular sclerosis or attached to this by a narrow neck, in which often lay a central vessel with walls changed according to the degree of sclerosis reached.

Horizontal sections through the hemispheres at Pierre Marie's *coupe d'élection*, or immediately above or below this level (Figs. 23, 24), showed that the occipital horn was surrounded by a hood of changed tissue, macroscopically greyish-white and gelatinous, or of a whitish-yellow colour and softer consistence. From the point of this hood the sclerosis is prolonged in a series of small elongated or rounded areas towards the posterior extremity of the occipital lobe, involving the optic radiations, the inferior longitudinal fasciculus, and the tapetum at several points, and in some cases involving the medullary rays and cortex of the convolutions of the calcarine fissure. The sclerosis of the anterior horns is less marked, but is also prolonged in the direction of the frontal lobe, and laterally involves the lenticular nucleus and the cortico-pontine fibres. Between these two horns the thickness of the sclerotic tissue varies greatly at individual levels—sometimes the lateral walls of the ventricle are scarcely involved, again there may be a sinuous narrow margin along its whole extent, and again the outer border at parts may be half a centimetre broad, with well-marked processes extending into the grey nuclei and adjoining white matter. The fornix and the corpus callosum, on the ventricular surface both of the splenium and genu, were also involved, and isolated areas were also found in both, which seemed to have no connection with the ventricular border.

Horizontal sections near the roof of the ventricle usually showed an involvement of the entire surface extent of the ventricular walls, with well-marked pouches passing inwards into the corpus callosum on the one side and the central white matter on the other (Fig. 26), and also passing upwards. One such area above the roof of the ventricle (Fig. 25), three-quarters of a centimetre in diameter, was traced not only to its connection with the roof sclerosis, but upwards till it gradually diminished and broke up into a series of smaller areas. In this large area there was one central vessel, but a large number of dilated vessels—arteries, veins, and capillaries—with their walls all filled with fat granule cells. The tissue was very soft and fell out in the large celloidin section; in the bichromate it had appeared chrome-yellow in colour, and much lighter than the surrounding white matter.

Horizontal sections through the temporo-sphenoidal lobe (Fig. 29) on both sides showed that the sclerosis of the descending horn was very marked, and below the floor of the ventricle it had extended

to involve almost the whole of the adjoining white matter, reached into almost every one of the medullary rays passing off from this, and that there were also numerous areas in the cortical grey matter in close relation to the affected medullary rays. Sagittal sections through the temporo-sphenoidal lobe in other cases gave a very instructive picture of the extension outwards of the ring of peri-ventricular sclerosis into each medullary ray, and of the very frequent involvement of the white matter of the hippocampal convolution, of the fimbria, and of the gyrus dentatus.

The ventricles, in most of the cases, were not dilated: their walls also were smooth, and presented neither granulations nor glandular indentations. In one case the lateral ventricles were very dilated, and the outline of the sclerosis between anterior and posterior horns was so irregular, and passed on both sides so deeply into the optic thalamus, in which were also numerous apparently isolated areas, that the substance of the optic thalami, the internal capsules, the lenticular nuclei, and external capsules presented a moth-eaten appearance. In one case, especially the numerous veins in the sub-ependymal glious tissue, especially near the posterior horns, were macroscopically outlined on the ventricular surface by a gelatinous, deeply-stained zone, and, microscopically, it was found that this corresponded to a zone of denser sclerosis—the vessels, however, in which had dilated adventitial sheaths filled with very numerous cellular elements. Only one distinct ependymal granulation (Fig. 285) could be found throughout the whole investigation: this consisted of a dense mass of deeply-staining glia nuclei and glia fibrils. The ependymal epithelium over it and over the whole sclerotic tissue in general, was retained and apparently normal—any apparent proliferation of epithelial cells could be traced to the oblique level of the section.

The histological structure of the peri-ventricular areas need not be entered into in any detail. Weigert sections showed the complete absence of myelin in the areas at all stages—the preserved nerve fibres passing into the areas were very tortuous and varicose. Marchi preparations (Fig. 138) again gave the long rows of fat granule cells in the tissue spaces and around all the blood-vessels, especially around the groups of venous vessels at both posterior and anterior horn: the branches of these vessels could be traced for long distances towards both occipital and frontal poles, and similarly numerous vessels could be traced into the lateral peri-ventricular

tissue. The closely arranged longitudinal fibres of the corpus callosum (Fig. 137) showed beautifully the tubular arrangement of the fat granule cells, the rows of enlarged spider cells, and the gradually increasing dense longitudinal fibril formation. Both Cajal's and Bielschowsky's methods and diffuse stains showed the very large number of retained axis cylinders. When the immediately subependymal tissue had reached the stage of dense sclerosis, it presented an extremely close fibrillar web, poor in nuclei—the fibres mostly parallel to each other, parallel to the direction of the normal nerve fibres. Numerous dilated vessels twined in this fibrillar tissue. Before the onset of this advanced sclerosis the glia meshes were often very elongated and rarified (Fig. 207); the glia cells were markedly drawn out in a longitudinal direction, and showed often a nucleus at each pole, and a sheaf of fine fibrils passing from each pole of the cell to interlace with similar bundles of fibrils.

In the zone of unaffected tissue around the ventricle it was found that there was frequently a proliferation of the subependymal glia cells. But such areas sometimes showed a lighter staining of the myelin: similar "shadow" areas often united wholly demyelinated areas of the lateral wall or extended inwards from them. It could thus be assumed that the originally primary areas became fused by these transition areas becoming wholly demyelinated, till the whole ventricular surface was affected.

Around the aqueduct of Sylvius and around the floor of the fourth ventricle and its lateral angles the degree of sclerosis was often very marked. The extent of this in individual cases is well brought out in the very numerous figures taken from Weigert sections, and is more fully described elsewhere in the individual cases. Its structure was everywhere on similar lines. The final web formed was simply an exaggeration of the closely arranged fibrils, which are normally oriented in all directions. The involvement of the cranial nuclei in this extension has been also elsewhere described.

4. Changes in Nerve Root and Meninges.

(a) Optic Nerve.

The optic nerve and olfactory lobe must, embryologically, be looked upon as parts of the central nervous system. They there-

fore contain neuroglia, and must be considered apart from the other cranial nerve roots. The olfactory lobe was examined in only two of the cases: both peduncle and lobe in each case showed distinct signs of demyelination. Owing to the extreme flattening of the tissue before embedding, it was found impossible to get very satisfactory preparations, and only Weigert and Van Gieson stained sections were examined.

The optic nerve, chiasma, and anterior portion of the tract, investigated in seven of the cases by numerous staining methods, in every instance showed marked involvement. In one case Weigert sections, both longitudinal and transverse, showed complete absence of myelin in the whole of the intra-cranial course of both optic nerves, and the chiasma was similarly degenerated. Figs. 64, 65; 261, 262; 274 show the degree of myelin involvement in six separate cases: the chiasma, as will be seen, was the site most frequently affected, and the first evidences were manifested in its anterior border. In one case the optic tract on both sides was cut in celloidin sections from the chiasma to the corpora geniculata interna, and both showed a very characteristic discontinuous degeneration: the degeneration in the chiasma passed into both optic tracts and then ceased—to begin again at a point midway in its course. Weigert counter-stained sections brought out very beautifully the advanced septal and blood-vessel change which accompanied the sclerosis in the optic nerves (Fig. 274). Van Gieson sections showed that this had commenced in a proliferation of the fine connective tissue elements between the nerve fibres, and that this active proliferative process had then extended to the larger septa and the inner layers of the optic sheaths. The glia elements had shared in this reaction, and there resulted rows of large protoplasmic glia cells with long branching processes. Later, the glia fibril formation seemed almost masked by the great connective tissue increase, which the very numerous blood-vessels of the septa had shared. The degeneration of the myelin sheath appears to take place very rapidly, and long tubular rows of fat granule cells are found in Marchi sections (Figs. 64, 65): the mode of their removal in the blood-vessel lymphatics is in every way comparable to that in the central nervous tissue. Redlich and others have noted the long persistence of the axis cylinders in the optic nerve sclerosis. In one case, macroscopically, there was very evident involvement of the optic chiasma and nerves: they

presented a completely gelatinous, almost transparent, appearance, and yet Cajal-stained sections showed an almost complete preservation of the axis cylinders in nerves, chiasma, and tract (Figs. 261, 262).

The characteristics of the areas in the optic nerves and chiasma are therefore related to the presence of both glia and connective tissue interstitial elements, for both are affected. The sclerotic areas in these regions are amongst the most constant appearances in disseminated sclerosis, and may be related to the early eye changes so frequently found clinically.

(b) *Changes in the Nerve Roots, Cauda Equina, and Peripheral Nerves.*

Changes in the nerve roots are comparatively seldom referred to in the literature of disseminated sclerosis, and where noted they are usually described as insignificant. Dinkler, however, in one case found the whole of the spinal cord roots thickened near the cord; Schob, in addition to fibroma-like thickenings of the nerve roots, describes discontinuous areas of myelin degeneration in non-glious containing tissue, and Strähuber and Marburg have reported similar findings. These areas contained a connective tissue proliferation of the endoneurium and a proliferation of the Schwann's sheath in place of a neuroglia sclerosis. In one peripheral nerve similar discontinuous areas were found.

In this investigation the nerve roots in only one case were systematically examined in longitudinal section, but in several other cases portions of the nerve roots remaining attached to the cord segments (Fig. 70) reached the length of one half centimetre, and in other cases very numerous ganglia were examined with nerve roots attached, and in most of the cases the cauda equina was examined in longitudinal sections extending from two to three centimetres. In the frequent neuroglial involvement of the intra-medullary portion of the cranial nerves, the extra-medullary portion seldom shared. The loss of myelin or the deficient staining of the myelin extended often for a short distance into the nerve roots, and then these resumed the normal staining. The extent of this demyelination and neuroglial involvement of the nerve roots varied greatly, and it was looked upon as proportionate in extent and form to the varying degree in which the glia

normally passed into the nerve roots. In the spinal nerve roots, however, especially in the posterior roots of the lumbar cord, this limit was frequently overstepped, and small circumscribed areas of neuroglial sclerosis were found, as if the glious zone had extended far into the normally non-glial portion of the root (Figs. 93, 94). Serial sections showed that these areas were very minute, and that the axis cylinders passed through them.

In addition to these small areas of sclerosis, there were frequently found in the fibres of both cranial and spinal nerve roots three other types of changes: (1) a diffuse change comparable to the "shadow" staining of the myelin in the central nervous tissue; (2) a definite secondary degeneration of isolated fibres or groups of fibres (Fig. 281), related probably to the loss of the axis cylinder in a sclerotic area; and, finally (3), in Marchi sections, as already mentioned, the nerve roots shared in an early degree of myelin degeneration, which affected the whole transverse section of many cord segments—a change which is probably related to the presence of a terminal infection. Orr and Rows have demonstrated the evidence of a continuous flow of lymph upwards along the nerves, the main current of which lies at the periphery of the nerve immediately under the fibrous sheath. It was presumable, therefore, that we ought to find in the cord of cases in which some septic focus existed, *e.g.*, bed-sores or an extensive pyelitis, lesions of the posterior columns, caused by toxins ascending in the lymph stream. The evidence for this was carefully sought for, but the lesions were already so extensive, and the terminal infection, if this were the cause of the general myelin degeneration, affected the whole transverse section, including the attached nerve roots, that no conclusion could be come to on this point.

Two points of interest may be added: (1) that the intra-medullary portions of both anterior and posterior nerve roots seemed frequently to withstand the sclerotic process much longer than the white matter through which they passed, and (2) Fig. 80 indicates how normal nerve roots may be attached to a segment of the cord entirely deprived of myelin, and Figs. 282, 283 that a similar condition of the nerve bundles of the cauda equina may exist through the whole lumbo-sacral cord shows, in Weigert sections, complete sclerosis. In both the longitudinal section and at every level of the cord in Case II., Bielschowsky prepara-

tions showed an almost normal number of retained axis cylinders. The posterior root ganglion related to numerous segments were investigated.

In only one case (L. W.) were the peripheral nerves examined. In both Marchi and Weigert preparations, both transverse and longitudinal, from two levels, at least 5 centimetres distant from each other, showed a complete absence of early or late degeneration in the following nerves: popliteal, peroneal, and median on both sides, and the right sciatic (Fig. 284). In the left sciatic nerve, however, there was a distinct Marchi degeneration (Fig. 154).

(c) *Changes in the Meninges.*

Borst has described, in all the four cases examined by him, adhesions and thickenings of the cerebral and spinal cord membranes. The significance ascribed by him to these changes is of great importance in relation to the pathogenesis of disseminated sclerosis, and this subject will be more fully dealt with when considering general changes in the lymphatics. A few histological details may, however, be given here. In uncomplicated cases the meninges were found almost normal; the pia was frequently slightly thickened, contained a slight increase of cells, and the pial vessels showed changes very similar to those within the sclerosed tissue. In the earlier stages the vessel walls and inner layers of the pia contained fat granule cells. In other cases, however, the cerebral and spinal soft meninges were infiltrated with cells, chiefly lymphocytes, and a few plasma cells, which also passed in with the vessels into the substance of the cord. Many dense glia fibres—radiating from the glia marginal zone into the pia—were found around the venous vessels as they passed out of the cord, and also around these vessels there was a marked accumulation of small round cells. These meningeal changes, when present, are usually diffuse and in no way confined to the meninges overlying areas of sclerosis.

(4) CHANGES IN THE INDIVIDUAL TISSUE ELEMENTS.

1. *Nerve Fibres.*

(a) *Medullated Sheath.*

In the actual sclerotic areas the medullated sheaths have entirely perished, but in recent areas and at the advancing

peripheral zone of older areas the changes present themselves in very various forms, according to the stages of the degenerative process, and according to the greater or lesser intensity of the process. In Weigert transverse section, the myelin sheath may appear deficiently stained, or there may be only a thinning of myelin, but more often it is diffusely stained and swollen, or the whole ring of myelin may be broken up into a number of finer and larger globules (Fig. 238). On longitudinal section, however, the process may be much more readily studied in its individual stages, and the fibres, especially in the transitional zone of an advancing area, may show all conceivable types (Figs. 236, 237). The varicosity so frequently found even in normal fibres is greatly exaggerated, and the whole fibre may be represented by a series of large oval vesicles, the outer rim of which stains with hæmatoxylin. Frequently, and this seems to me the most usual type, a number of very fine granules and balls are attached to the outer border of the myelin sheath; some of these tiny balls seem to burst, and gradually others reform till the myelin sheath projecting into an area is gradually more and more thinned, and is finally represented by a series of very delicate globules. Some of these may be found for a considerable time in the degenerated area, retaining the hæmatoxylin stain. One rarely meets with the very swollen, tumefied, badly-staining fibres such as are found in softenings, although the destruction of the myelin seems to start in an œdematous swelling analogous to that found in acute myelitis.

In Marchi sections (Figs. 149-153) the actual degeneration shows first as long parallel rows of droplets which darken with the osmic acid. These are again most frequently on the outer edge of the nerve fibre, and may be only on its one side. Such fibres on transverse section would appear as if a crescentic part of the sheath were affected. Sometimes there are double rows of droplets or granules within one myelin sheath. The appearance of these rows of granules is often, but not always, preceded by a varicose condition of the fibres, extending over long distances; the swelling affecting the whole sheath and appearing either in the form of small beads or large spindle-shaped vesicles, which may perhaps represent an acuter process, or one accompanied by more œdema. Within these swellings the sheath may then show the fine granular degeneration, and these granules may run together. On transverse

Marchi sections it is frequently possible to recognise that an entire outer rim of the sheath is stained black, and that this degeneration gradually extends inwards to involve its whole extent.

In Heidenhain's iron-hæmatoxylin sections, the nerve-fibres bordering on the degenerated zone have their neurokeratin framework beautifully brought out, and in the spindle-shaped swelling above referred to this is seen to be a very wide-meshed one. Marburg has represented the chemistry of this process as a degeneration first of the lecithin, which supplies the fatty products, while the chief mass of the hæmatoxylin substance (protagon) is spared longer, so the fibres which in Marchi sections show degeneration are still stained in Weigert's hæmatoxylin.

In longitudinal sections of the cord it may be seen that this degeneration may affect the nerve fibres "discontinuously." This is specially easily recognised in Marchi sections, counter-stained with safranin, in which the retained axis cylinder is seen surrounded by a pink-stained zone, which becomes gradually interrupted by a portion of the fibre in which granular disintegration has commenced. The axis cylinder within this affected portion may be swollen and homogeneous, but can be recognised till a late stage of the degeneration. Thus this form of degeneration can be distinguished from secondary degeneration by its limitation, by its late affection of the axis cylinder, and, finally, by the character of its disintegration into granules and globules (Fig. 150) instead of at once into coarse balls and fragments (Fig. 154) which fill the whole myelin sheath. The fat granule cells, which are found in such large numbers, could only in rare instances be found to contain large fragments of myelin. It seemed rather as if a gradual absorption of dissolved substances had taken place, and that these within the cell had become transformed into fine fat-like granules.

In the "Markschattenherde" we may have simply a deficient staining of the whole myelin sheath, or there may be only a thin ring of myelin around an almost normal axis cylinder. Volsch assumes that in the latter case there has been a gradual atrophy of the myelin, and then a persisting condition of the remainder.

It may finally be noted that in some areas certain fibre-systems seem to be more resistant. In areas of sclerosis involving both the anterior horn and the entire antero-lateral tract the intra-medullary anterior root fibres were frequently found intact, or

showing only slight Marchi degeneration. Similarly the external arcuate fibres in the medulla oblongata seem to be relatively long preserved.

(b) *Axis Cylinder.*

The comparative persistence of the axis cylinders in the sclerotic area has long been regarded as one of the essential characteristics of disseminated sclerosis. Charcot himself, from the apparent disproportion between the symptoms and the anatomical lesions, argued for their persistence. The earlier observers undoubtedly, with diffuse stains, frequently mistook the longitudinal glia fibrils, which run parallel to the degenerated nerve fibres, for true axis cylinders, yet modern specific staining methods have proved that Charcot's conception was justified. Yet this persistence is only a relative one, and in the foregoing study attention has been drawn to the numerous alterations which axis cylinders undergo. The most frequent change is that of a slight homogeneous swelling, and this may be found even in old sclerotic areas, in which, as a rule, the axis cylinder becomes attenuated, and, with diffuse stains, is only with difficulty distinguished from the coarser fibrils. The changes in the axis cylinders are much more easily recognised in longitudinal sections of the nerve fibres.

In early areas degenerative changes (Figs. 255 and 256) set in with a moniliform appearance, which may later take the form of spindle-like enlargements of various size, and these may finally lead to disintegration, with the formation of homogeneous clumps and granules. This qualitative change may affect a large number of the axis cylinders in an affected area, or most of the fibres may show spindle-shaped swellings and only a few go on to the stage of disintegration. It is probable that the fibres which survive the swelling may persist into the stage of sclerosis as homogeneous, condensed elements which, as the sclerosis becomes denser, are compressed into thin, even spiral threads.

In the stage of abundant fat granule cell formation the swollen axis cylinders become pushed aside between these cells and the proliferated glia elements (Figs. 1, 2; 158, 159), but with specific stains it can be seen that as these cell elements diminish in number and size, the axis cylinders course straight again and are surrounded, almost as by a sheath, with the proliferating, wavy, glia fibrils (Figs. 3; 154). At the margin of a demyelinated and even sclerotic tissue,

the direct transition of the still remaining axis cylinders into those of the normal tissue can be followed, and if the area be not a very long elongated one, a normal myelinated axis cylinder can be traced, deprived of myelin, right through the sclerosis tissue to its transition into a normal myelinated axis cylinder again. In other cases the transition from normal tissue into sclerotic is marked by a fainter grey-staining of the axis cylinder, which in the depth of the area becomes a mere shadow (Fig. 253), or the transition may be represented by a swelling and tortuosity, which it must be remembered are present to a limited extent in quite normal tissue. If the axis cylinder, too, terminate at the transitional zone, their ends are often swollen and granular. On transverse section the remains of such degenerated axis cylinders may be recognised as faintly-staining granules—with eosin or picro-fuchsin—or dark-stained, cloudy granular debris—iron and hæmatoxylin and silver—lying in the meshes of the glia cell protoplasmic processes, and in the walls of the blood-vessels.

The density of the non-myelinated axis cylinders is rarely the same (Fig. 252) as that of the normal fibres, but sometimes they are found of normal calibre and numbers. A striking illustration of this may be seen in Figs. 16, 17; 251. In considering the relative proportion of axis cylinders preserved, it must be taken into account that the sclerosed area is relatively smaller than normal, *e.g.*, where the sclerosis affects the entire transverse section of the cord the area is frequently little more than half the normal. In this very shrunken area impregnation methods show a dense arrangement of the axis cylinders, yet one must admit that many must have perished.

Numerous writers have claimed that after the degeneration of the axis cylinders there comes a regeneration. Popoff, Marinesco, and Minea, and others base their contention on the presence of axis cylinders with a brush-like formation of fine fibres at their end, or on the presence of *boules terminales* at the end of both terminal and collateral fibres. This possible regeneration has been supported by Schmaus and Huber, and also by Strähuber, on the strength of his aniline-blue staining methods. Bielschowsky, while claiming that most of the fibres in a sclerotic area are "persistent," admits the possibility of a regeneration. Borst, however, who has investigated the regenerative powers of fibres in the brain, and ascribes to them a very considerable regenerative new formation,

thinks that both Bielschowsky's and Strähuber's special methods stain certain kinds of glia fibrils so similar to axis cylinders that it is impossible to draw any conclusion.

Marburg claims that the first effect of the "toxine" is a lecitholysis: when this effect is carried further or is more intense there is a marked axolysis with the formation of fine granules. But it must be admitted that a change short of this, the demyelination and the swelling of the axis cylinder—which is an indication of its sensitiveness and a proof of its sharing in the changes in the myelin sheath—may be present.

2. *Nerve Cells.*

The relative resistance ascribed to the axis cylinders has also been extended to the ganglion cells, both in the cord and brain. Numerous writers have emphasised their quite normal appearance, both in outer form and minute structure, till a late stage of the process. Catolà found normal cells even when sclerosis was complete, and Schlagenhauser, in a case of disseminated sclerosis which ran its course as a transverse myelitis, found the nerve cells normal at all levels of the cord, even in the most affected segments.

The relation of the cell changes, when present, to the sclerotic process is difficult to decide, and the majority of the changes should probably be referred to the intercurrent disease, with possible elevation of temperature, which caused death, or to the exhaustion and anæmia and decubitus which accompanied the nervous affection. In judging how seriously diseased a cell is, and specially as to whether it is capable of restoration, the variations in the nucleus have always been counted as affording more useful data than those of the protoplasm. In one case (C. S.), in which every level of the cord showed not only an almost complete demyelination, but cell and glia stains showed that the fat granule cells had been almost completely removed from the tissue, and already an advanced fibril formation was in process, many ganglion cells at very numerous levels retained an almost normal nuclear structure and an almost complete retention of the chromatophile granules. (Fig. 240). The only recognisable change was an absence of the processes, a rounding of the cell body, and an increase in the cell pigment. At other levels, all the cells present showed a marked diminution in volume (Fig. 239), and a marked pigmentation (Fig.

243), including cells which had become already transformed into a rounded, small, non-nucleated mass of pigment. These changes occur in areas in which the sclerosis is not very advanced, and the intercellular tissue is composed of a network of capillaries and branching glia cells. In a later stage such cells become wholly lost in the sclerotic tissue, or small traces of pigment may still be found.

Another type of change, and a more frequent one, is a gradually advancing atrophy (Figs. 245, 246), proportionate to the increasing density of the sclerosis. This first affects the minute structure of the processes, and then reaches the cell body, in which the chromatophile granules become powdery or are dissolved, and the nucleus becomes peripheral. Such atrophying cells may assume very varied shapes according to the intensity of the process, and traces of non-nucleated cells with complete chromatolysis may be found in the sclerotic tissue for a long time. In the cells of Clark's column, where we have normally an excentric nucleus and a peripheric disposition of the chromatophile granules, the nucleus undergoes a marked shrivelling and condensation, or at times a vacuolation before its extrusion. The cells very frequently assume a spindle shape, and have been described by Nissl as "Fisch" cells.

The mechanism of these changes in the spinal cord cells seems to be a simple atrophy, and one factor in its causation is probably compression on the part of the developing cells and fibres of the glia (Fig. 249). Nowhere was there found any evidence of the accumulation of small round cells around ganglion cells in the cord, such as are to be described later around the ganglion cells of the cortex. The changes described are those related to a slow, chronic process, but more extensive and general changes, *e.g.*, complete chromatolysis (Fig. 241) or deeply-staining cytoplasm, are also found which must be related to the general somatic disturbances. The "coagulation necrosis," described by Marinesco as an acute cellular change in which there is a dense fusing together and coagulation of the individual constituent elements, is rarely present.

The cells in the different cranial nuclei undergo changes which are closely analogous to those described above. The cells of the hypoglossal nuclei are frequently very pigmented, and have the pigment distributed throughout the whole cell. The glia cell proliferation may be very marked before there is any appreciable atrophy of the cells, but as the sclerosis advances, here, too, there is a gradual diminution in their number (Fig. 248). Cells with

normally staining granules may be found alongside cell remnants or pigment accumulations.

The cells in the cortex, in a demyelinated area, when involved, are much more uniformly so than those of the cord. The large pyramidal cells are never normal (Fig. 216), but show chromatolysis and have their dendrites thickened or weakly-stained or absent, and the cell contour is rounded or pear-shaped (Fig. 22). These changes involve not only the demyelinated area, but also the adjoining stretches of the cortex to a varying extent. But the most characteristic appearance in a cortical area, and that which enables it, under low power, to be readily recognised, is the nuclear increase around the ganglion cells. The majority of the ganglion cells, in the lower layers of the cortex and in the layer of the large pyramids, are surrounded by small nests of cells (Figs. 211, 213), from five to ten in number. These with diffuse stains have a small deeply-staining nucleus with little protoplasm, and cause, according to their number and arrangement, more or less deformity and atrophy of the ganglion cell, but we have never seen the penetration of such cells into the ganglion cell body. They have probably arisen from the pre-existing satellite cells, and their proliferation is taken as an indication of the inter-relation between ganglion cell and its satellite cells ("Trabantzellen"). Marinesco thinks that in health the former secrete substances which exercise a controlling influence on the size and development of the latter; if any noxious or toxic agent affects the former, this controlling secretion is diminished in quantity and quality, and the neuroglia cells react accordingly by increasing in size and numbers. This change is again not limited to the demyelinated areas, but passes over to the adjoining stretches of the cortex.

Comparatively little change was found in the cells of the posterior root ganglia (Fig. 250). Those that were present indicated a general reaction, probably in no way related to the sclerotic process in the cord.

3. *Neuroglia.*

The changes in the neuroglia represent, in the opinion of numerous observers, the dominant and essential histological feature of disseminated sclerosis. Whilst the changes in the nerve fibres

show little variation in the individual cases, the glia components show wide differences. In an early area we have seen that the most important and characteristic finding is the enormous number of large cell elements (Fig. 210), which in their further evolution are transformed into glia fibrils (Fig. 214) and glia nuclei. In an actual sclerotic area we have a dense glia fibril mass, apparently without spaces (Figs. 184, 194). The histological characters of the glia, therefore, depend on the length and the intensity of the process. In the final result the name "sclerosis" is justified by the amount of neuroglia present in the areas, and Weigert has stated that the glia hyperplasia is greater here than in any other form of sclerosis. The neuroglia, though not from a histogenetic, yet from a morphological and biological standpoint, may be looked upon as a true fibrous connective tissue. It is thus of great significance that the glia begins to proliferate when component parts of the specific nervous tissue are destroyed, even where any stimulus capable of causing primary proliferation is absent. The classical example of this is the secondary degeneration of the white substance, where the secondary glia proliferation sets in probably as a reaction to the irritation caused by the products of degeneration. At the same time it may happen that one and the same "noxa" destroys the specific nerve tissue, and in an equally primary manner causes the glia to proliferate. Again it is possible that we may have isolated primary proliferative processes in the glia—which in their turn cause secondary degeneration of nerve cells and fibres. Storch has pointed out that in chronic diseases, in which the plan of the nerve tissue remains unchanged, the newly-formed glia fibrils show exactly the same arrangement as the original fibres, whereas in cases of acute destruction of tissue, this regularity does not hold good. He, therefore, distinguishes between an isomorphous and a reparatory sclerosis. The glia proliferation may, therefore, be merely a substitution process, or an inflammatory process, or, more rarely, a primary glia proliferation.

It is impossible to discuss here the question of the spatial relation of the glia fibres to the glia cell protoplasm. All that can be done is to indicate the stages in the elaboration of the glia fibrils (Figs. 212-214). These can be followed very beautifully in the evolution of an early area into a sclerotic area by means of Heidenhain's iron-haematoxylin method and Ford-Robertson's

methyl-violet stain. In the rapidly proliferating glia cells, the first stage in their transformation is an enlargement of the nucleus (Fig. 8), by which its chromatin structure becomes clearer. This is followed by the development of a considerable amount of deeply-staining protoplasm around the nucleus, and the further development of large, branching, protoplasmic processes (Figs. 9, 179), till forms of very varying size and shape are produced. In many of these large glia cells, two or more nuclei may be found (Fig. 209): this may represent a karyokinesis which has remained incomplete. After cell division the new cells rapidly increase in size, and form protoplasmic processes; and are potential fibril-forming cells (Figs. 209, 210). From a close consideration of the specimens the conclusion was reached that the formation of fibrils can take place in the enlarged pre-existing glia cells without cell division. The first indication of the formation of fibrils consists in a definition of the edge of the protoplasmic processes (Fig. 212); when this can be followed throughout the concave border of two adjoining processes (Fig. 213), it gives to the fibril formation the appearance of recurving fibres (Fig. 214) with their convexity near the cell nucleus. The general arrangement of the fibrils corresponds at first to the general outline of the borders of the protoplasmic processes. At a later stage the relation to individual nuclei is less easy to determine. Ford Robertson thinks that each branching process becomes converted into several plain processes by gradual splitting at the forks down to the close vicinity of the nucleus. His methyl-violet method shows very clearly that many of the fibres are attached to the walls of a vessel by an expanded "foot," but frequently, especially around the capillaries in the cortex, the new formed fibrils of adjacent cells were found to form a network, with elongated meshes, around the capillary wall (Figs. 21, 221).

Glia cells, that have produced fibrils, undergo slow and gradually regressive changes: almost the whole of the protoplasm seems used up and shrivels into an irregular border around the doubly-staining nucleus. In later stages, too, the nuclei may disappear, and thus account for the comparatively few nuclei found as a rule in actual sclerotic areas.

Weigert's method does not stain the cell body, and therefore his statement as to the spatial separation of the glia fibrils from the cell protoplasm may be overdrawn, but this does not under-

mine the "Verhalten," the distribution, and the specificity of the fibrils, and the fact that they stain at a certain definite stage of their development. Fieandt's recent work has seemed to support the Hardesty-Held conception of the syncytial structure of the glia tissue. Held believes that the glia represents a widely-ramified but connected syncytial meshwork of protoplasmic character, which envelops the functioning elements of the central nervous system. At its nodal points are nuclei, and within the protoplasm, not separated from it, are Weigert's specific fibrils. The glia, therefore, represents a continuous reticulum, which holds together the ganglion cells and nerve fibres, and the differentiated fibrils are deposited in this as supporting or stiffening elements. By Nissl's stain the cell nucleus and protoplasm immediately surrounding it are stained, and the cell protoplasm gradually merges in a surrounding meshwork. The sharp concave edges of the bodies are formed by light, strongly refractive lines, which consist of fibrils that do not stain with basic aniline dyes. Fieandt thinks that glia granules—gliosomes—play an important rôle in the new formation of glia fibrils, and that they may be looked upon as an intermediate stage between the undifferentiated protoplasm and the specific fibres. In specimens fixed in Heidenhain's sublimat-trichloroacetic acid mixture, and stained by iron-hæmatoxylin, granules were present, arranged in rows, in the large star-shaped glia cell processes and in different parts of the cell body. In some cases rows of granules are seen to radiate towards the centre of the cell, thus giving the cytoplasm a radial structure. These granules are very fine, and similar granules may be distinguished in the fine meshes of the glia, arranged almost in streptococci-like rows.

The Hardesty-Held conception of the syncytial structure of the glia tissue would reconcile Weigert's teaching with that of his opponents, and show that in the neuroglia tissue cells—nuclei and protoplasm and protoplasmic processes—differentiated fibres, whether anatomically independent or not, and, finally, intercellular protoplasmic fibreless glia may all exist together.

In the grey matter of the brain the ganglion cells and fibres are embedded in a tissue which shows a uniform finely granular structure. This is probably constituted by the dendrites and axis cylinder processes of the ganglion cells; the axis cylinder ramifications from other parts of the grey matter; the fine intercellular

fibril lattice work originating from both; and by the diffuse protoplasmic glia meshwork with its specific glia fibrils. In this glia meshwork nuclei are fairly evenly distributed, and are specially met with in close relation to the larger ganglion cells—the so-called satellite cells or “Trabantzellen”—and around the vessels. These elements, together with arteries, veins, and a fine capillary network, constitute the grey matter of the cortex. The specific glia fibrils vary much in amount in the different layers. In the marginal zone and amongst the tangential fibres they are abundant, but from this downwards they rapidly decrease in amount, but in the transition between white and grey matter they are again abundant. On the other hand, the protoplasmic glia meshwork is apparently equally developed at all levels. Radial glia septa, such as are found in the cord, are never present in the cortex. The nuclei in the grey cortex are small and round, 5 to 7 μ in diameter, or flattened, and have a dense chromatin content in the form of fine granules. Larger and lighter-stained nuclei are also met with, and both types have, with certain stains, fine thread-like processes, which ramify and anastomose with each other.

We have stated that one portion of the proliferated glia cells, both in the white and grey matter, probably form the first fat granule cells. This double function of the neuroglia cells, that of fibre-formation and phagocytosis, has been emphasised by Nissl, Marinesco, and Ford-Robertson. The increasing zone of protoplasm around the nucleus becomes gradually laden with granules, which often outline the cell body and its processes—at first spindle-shaped, then star-shaped, and gradually becoming rounder till the cells are set free in the glia reticulum (Figs. 18-20). These glious granular cells have a lattice or “Gitter” structure in their protoplasm, in preparations in which the fat granules have been dissolved out (Figs. 9, 10). They emigrate from the tissue, or rather are carried along with the lymph stream, and are found later in the adventitial spaces of the blood-vessels, and there give up their contents or are carried still further on. This evolution may be traced beautifully in the advancing zone of an area in the white matter where the nerve fibres are cut longitudinally. Here the pre-existing small glia cells, lying far into the normal myelinated tissue, may be found proliferating and undergoing the changes described above. In the cortical grey matter this evolution is

slower, and the commencing granule formation in the "Trabanzellen" is well brought out in Fig. 226.

4. *Blood-vessels; Lymphatics; Cell Elements in Vessel Walls.*

(a) *Blood-vessels.*—Note on the normal structure of the vessels of the central nervous system.

The arterial vessels of the cerebral cortex arise altogether from the pia. Short radial branches, referred to as "cortical" vessels, pass into it perpendicular to the surface, and soon after their entrance break up into the very smallest arterioles and capillaries. The outermost layers of the cortex are supplied, not by lateral branches from the larger of those radial arteries, but by a special system of very short vessels, which break up into a capillary network in the first and partly in the second cortical layers. One division—of longer radial branches, referred to as medullary—penetrates further down and supplies the superficial portion of the subcortical white matter. The larger portion of the cortical white matter and the basal ganglia are supplied by central arteries, which arise from the circle of Willis; their terminal branches ramify on the surface of the ventricles (Borst). One division of the veins likewise passes to the pia, and another, that draining the central white matter and basal ganglia, joins the great veins of Galen beneath the splenium of the corpus callosum. Numerous venous branches can be recognised, immediately sub-ependymal, coursing mostly obliquely towards this point, and grouped especially around the posterior horn of the lateral ventricle. In the spinal cord the white columns are supplied by vessels radiating from the pia, and the grey matter by vessels passing in from the anterior fissure. The transition zone between white and grey matter receives its blood-supply from the terminal branches of both central and lateral vessels. The lateral vessels, as they penetrate the cord, run at first almost in the transverse section of the cord; many lateral transverse branches are given off, but the majority widen out in vertical directions. These vertical branches, on account of their small calibre, represent terminal vessels, pre-capillary arterioles, and capillaries, and may be traced, in longitudinal section, for long distances. In consequence of this arrangement, the areas supplied by the radial vessels and their lateral branches would, therefore, have a wedge-shaped form,

with base on the surface of the cord, and those supplied by the vertical branches must have a shape in the long axis of the cord.

The discrimination, in individual sections, of small arteries and veins is by no means always easily made, and, when a condensation of the vessel wall occurs in sclerotic tissue, is impossible. As in other tissues the venous walls are thinner, contain irregularly distributed muscle cells and adventitial nuclei, but small veins proceeding from capillaries cannot be distinguished from pre-capillary arterioles. It is generally taught that no elastic lamina is present in the arteries and arterioles, but Schroeder and Lapinsky state that a very delicate elastic membrane can be recognised even in the capillaries, and that elastic fibres are present in the media and the adventitia, wherever a distinct adventitia can be found. With Weigert's elastic stain the capillary walls certainly stain sharply and frequently with a double contour, which some have looked upon as the expression of a rudimentary elastic membrane. It is generally admitted that even the capillaries have traces of an adventitia, the nuclei of which are found only at intervals. The capillaries, therefore, consist of an endothelium and traces both of an elastica and an adventitia. In pre-capillary arterioles we get, in addition, detached muscle cells, which in the larger arterioles form a layer of circular cells. Ford-Robertson states that by far the most important feature in the structure of the intracerebral vessels, in relation to the physiology of the cerebral circulation, is the remarkable development and the highly elastic character of their adventitia, which invest not only the arterioles but also venules and capillaries. The capillaries of the central nervous system, therefore, unlike the capillaries of most other organs, possess an adventitia, the fibres of which differ both from white fibrous tissue and yellow elastic tissue. He thinks that there is strong evidence that in various toxic conditions the capillaries of the central nervous system are prone to undergo definite change, while those of other organs escape. The explanation of this selective action is to be found in the fact of their higher structural differentiation, which has been obtained at the cost of a peculiar vulnerability to the action of certain toxins.

The adventitia is immediately bounded on its outer side, without the presence of any peri-vascular space, by a condensed layer of glia—the glia peri-vascularis limitans. This glia layer is

a continuation inwards of the glia superficialis limitans which was carried inwards by the ingrowing vessels of the pia when they invaginate the embryonic nerve tube. The inner layers of the pia form the adventitia coat of these penetrating vessels, and this intimate relation between glia and adventitia is maintained till the finest capillaries are reached. In these the glia limitans may be formed by a very fine protoplasmic reticulum in which the glia "Fuss" of immediately adjoining cells are inserted.

It was formerly supposed that there was no evidence of the existence of vasomotor nerves in the intracerebral arteries, but Lapinsky has demonstrated that medullated fibres reach the media and are distributed to the muscular fibres as non-medullated fibres.

The disposition of the areas in relation to the blood-vessels has led to the supposition that these play an important rôle in the genesis of the areas. This topographical relation is frequently obvious even macroscopically, especially in areas in the cerebral white matter, and numerous illustrations point to its microscopic proof. This dependence on the vessels is brought to light only where the areas are isolated; in later stages, through coalescence, the original relationship is no longer recognisable. A parallelism between the sclerosis and the area of distribution of a vessel would argue that the development of the process depends upon the condition of the vessel or of the "noxa" circulating within it.

To the alterations in the vessel walls in sclerotic areas numerous writers have, therefore, ascribed an essential significance, while others have regarded them as accessory and subordinate. Few writers have noted their entire absence; Taylor, in eight cases, could find no trace of vessel change, and Erben, in a careful investigation of five cases, found none that were not common to the whole central nervous system. Variations in the findings have been explained by the different stages in the development of the sclerotic process. In early stages engorgement and dilatation, cell infiltration of the walls, dilatation of the lymphatic sheaths, and capillary hæmorrhages have all been noted. At a later stage, marked nuclei increase in the adventitia and, in the actual sclerotic areas, condensation and hyaline change of the vessel walls. Probably none of the histological changes have been so variously interpreted as those related to the vessels.

The sequence of the changes in the blood-vessels seems to me

to be the following: at an early stage the blood-vessels in an involved area become dilated and engorged with blood. It is not a question so much of one central vessel as of all the branches of one small vessel, and even the finest capillaries, are recognised; the vessels are in no way altered, and have no nuclear increase either in the intima or adventitia. During the stage of marked glia-cell proliferation and commencing fat granule cell formation, in very numerous areas it has been impossible to recognise any structural alteration. The impression of a new formation of blood-vessels, both at this stage and at a much later one, is probably due to the engorgement making each fine vessel stand out distinctly, especially when stained with picro-fuchsin. Nowhere at this stage is the relation of individual parts of the tissues altered. In a few cases there have been noted a proliferation of the capillary endothelium, and the possible passage outwards of the proliferated endothelial cells into the tissues. At the stage of abundant fat granule cell formation, when these cell elements are passing into the lymph spaces of the adventitia, every vessel in the affected zone is mapped out by a ring or rings of such cells (Figs. 10, 13). The smallest capillaries show a single row of cells (Fig. 263), often with an outer limiting membrane, stained pink with fuchsin or blue with Mallory's connective tissue stain; the presence of this outer limiting membrane to such a row of cells seems a strong argument in favour of the existence of a thin adventitia in the capillaries. If such an area, with fat granule cells crowding the vessel sheaths and tissue spaces (Fig. 264), be looked at, with low power, and especially in celloidin sections, where it is more difficult to analyse the constituent elements, the impression is given of a softened area with cell-infiltrated walls. The possibility that areas at such a stage of development have been taken as illustrating cell-infiltrated areas may explain the great significance that has been ascribed to the vessels and to the inflammatory character of the process. This is still more evident at a slightly later stage, when there is a reaction to the presence of these fat granule cells in the adventitial spaces, and a proliferation of their cell elements. The cell proliferation is of a secondary nature, and is evident in uncomplicated cases we think, only at this stage. It is evident that there is a marked increase in the nuclear content of the vessel walls, an increase in which the endothelium of the capillaries shares. As the resorption processes

advance there is a gradual removal of the fat granule cell element in the adventitial spaces, but the cell body of many of these cells has gradually disappeared *in situ*, leaving a deeply-stained, crenated nucleus (Fig. 14), and during this stage we get a further nuclear element added to the nuclear increase in the vessel wall. The adventitia has had its fibrils dissociated and its lymph spaces filled by the fat granule cells; as these disappear the lymph spaces, which remain distended for a considerable time during the advancing sclerosis, are to a certain extent now occupied by a cell infiltration of another kind—these are the small, round, lymphocyte-like cells common to all chronic processes. These, with the elements distinctly recognisable as proliferated from the endothelium of the adventitial spaces and from the adventitial connective-tissue cells, together with a few remaining fat granule cells or their nuclei, form the cellular content of the vessel walls at this stage. Figs. 14 and 269 show beautifully the varieties of the cells found in such a vessel. I have never seen any marked grouping of small round cells analogous to the so-called “round cell infiltrations.” As the sclerosis advances the dissociated fibres of the adventitia gradually come together, and proportionate to this is a gradual diminution in the contained cell elements (Figs. 15 and 270). For a long time the outer adventitial fibres form a reticulum which can scarcely be distinguished from the peri-vascular glia reticulum, and in the meshes are still found various cell elements. At a still later stage the sclerosis affects all the parts of the vessel wall, and the dissociated fibrils form a fused, homogeneous, almost hyaline layer. As the vessel becomes hyaline, the elastica becomes broken up and lost, so that nothing can be recognised of specific muscle, connective tissue, or elastic elements (Figs. 270, 271). The relation of the proliferating glia cells to the small vessels has already been emphasised, and at the later sclerotic stage the proliferating fibrils, perpendicular to the vessel wall, form almost a radiating ring around the vessel; but very frequently, especially in cerebral areas, the new-formed glia fibrils are laid down in concentric rings around the vessels (Fig. 267). It has also been mentioned, but may here be emphasised, that it is frequently the peri-capillary glious zone in which the first traces of glia proliferation are noted.

In the majority of the sclerotic areas all the vessels are con-

densed—arteries, veins, and capillaries—and it is not possible to draw any distinction between arteries and veins, arterioles and venules (Figs. 272-274). Similar condensation of the vessels is found in all chronic sclerotic conditions in the nervous tissues, and Lugaro has pointed out that this is in keeping with the small claims of their nutritive function, for the sclerotic tissue requires less nourishing material.

(b) *Lymphatics.*

The increasing recognition of the importance of the lymphatic circulation in the central nervous system has led to numerous researches upon the precise nature of the lymph paths and the direction of the flow of the lymph within them. It cannot be said, however, that any exact knowledge on either of these points has yet been revealed. The present section of this paper is confined to the indications of changes in the lymphatic vessel system in disseminated sclerosis. The lymph apparatus shares in the changes in the blood-vessels, and Borst has strongly urged the view that a disturbance in the lymphatic circulation is the essential factor in the process underlying disseminated sclerosis. He points out that the factors which bring about this disturbed circulation are alterations in the vessel walls and in the meninges. Even slight affections of the former involve changes in the transudation of the lymph and lead to pathological exudations, and even slight adhesion of the pia to the marginal glia zone would involve occlusion of the epi-spinal or epi-cerebral spaces, an occlusion which would be extended to the peri-vascular spaces at the periphery, and therefore cause a hindrance to the outflow of the lymph by these channels. In a recent paper the late Dr Bruce and I indicated our agreement with Nissl's contention that the adventitial lymph spaces of the blood-vessels, which open into the spaces of the inner layers of the pia, are the only true lymph spaces of the central nervous system. It has seemed, therefore, that this assumption of epi-spinal, epi-cerebral, and peri-vascular spaces is an error which invalidates greatly Borst's views, and causes him to overestimate the part played by these adhesive changes. The fundamental importance of an increased transudation of toxic lymph cannot be overestimated, but the evidence of a hyperlymphosis is not by any means clear except in isolated cases, and its explanation when present cannot always be traced

to be secondary to a closure of adventitial spaces. Borst thought that together with the change in the vessel wall, which allowed increased transudation, there was a closure of these adventitial lymph spaces, so that the lymph, unable to get back, dilates the tissue interstices, especially the peri-vascular glia and the peri-vascular space. In these dilated glia meshes the first changes of the nerve fibres occurred, and later, secondary to the degeneration, there was a substitutive proliferation in the glia. Such areas with dilated meshes he termed "Lichtungsbezirke."

In the course of this study attention has been directed to the fact that the adventitial lymph spaces showed in the early stages only a slight degree of dilatation, that, later, they were distended by the presence of fat granule cells, and that they remained dilated and filled with cell elements of various kinds till a late stage of sclerosis. In the early stages there was no question of a primary proliferation such as might be induced by a toxic lymph flowing in them—the cell elements of the adventitia are, firstly, the fat granule cells, secondly, these, together with proliferated cells of the adventitial walls—a result of the reaction to the foreign elements in the spaces—and, finally, a cell infiltration of lymphocyte-like cells. In an actual sclerotic area the adventitial lymph spaces were closed in the general condensation of the vessel wall, but here the relations of the lymph in the narrow interstices of the dense glia tissue must be very different from those in the normal tissue. The areolar zone, which is sometimes found around areas of dense sclerosis, may be due in part to the obstruction of the lymph flow from normal tissue into sclerotic tissue.

Very numerous peri-vascular sieve-like areas (Figs. 278-280) were found, but these were frequently related to normally myelinated tissue. The change consisted in an extreme dissociation of all the fibres in the adventitia, so that the space between media and the peri-vascular glious zone was occupied by a connective-tissue reticulum with widened meshes which contained cell elements and frequently a granular deposit. There were frequently also found indications of a lymph congestion which extended over the whole transverse section of the cord, and which found expression in dilated adventitial lymph spaces, distended glia meshes, and a swelling of the contained myelin sheath and axis cylinder. In uncomplicated cases only occasional and insignificant variations

in the soft meninges were present, yet it must be admitted that changes, if minute, might readily escape observation.

(c) *Cell Elements in the Vessel Walls.*

The statement is frequently made that infiltrations are met with in recent areas, and such cell infiltrations of the adventitial sheath are considered characteristic of the inflammatory process in the nervous tissue. In numerous references to the cell elements of the adventitia, an endeavour has been made to show that the first cell infiltration is one associated with resorption processes (Fig. 13); that this is followed by a cell proliferation in the adventitia, which is probably a secondary reaction process to the presence of the first cells in the lymph spaces (Fig. 267); and that, finally, we have a more or less marked degree of cell infiltration which is characteristic of a chronic inflammatory process in any tissue (Fig. 14).

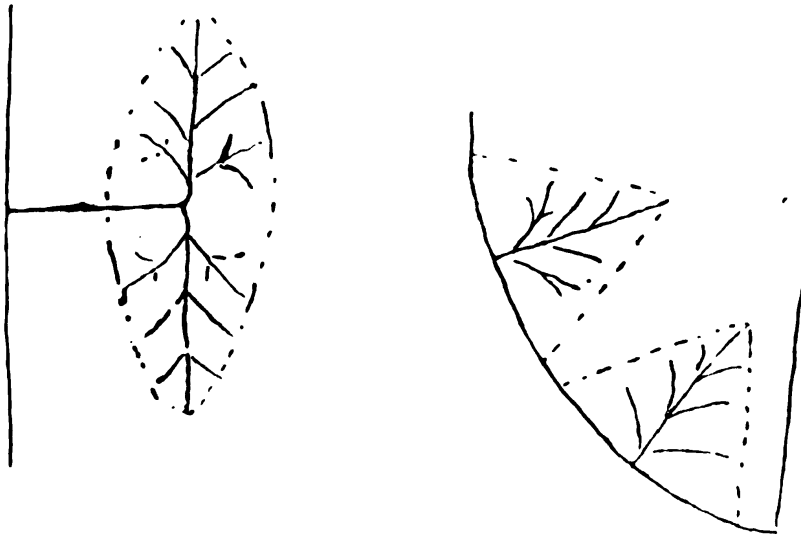
In the earliest stage, as a rule, there is no evident increase of the nuclei in the vessel wall, but sometimes a slight proliferation of capillary endothelial nuclei and adventitial nuclei is found, which may be related to the production of fat granule cells. In the stage of secondary reaction to the presence of the fat granule cells, the resultant cells in the adventitia are of two kinds; the one, with large nucleus and clear chromatin framework, have probably arisen from the endothelial nuclei of the lymph spaces, the other, with darker-stained nuclei, from the proliferation of the connective-tissue elements of the adventitia. In the third stage, in which the cell infiltration partakes of the characters of a chronic process, we have mostly lymphocyte-like cells, with a darker nucleus than any of those mentioned above, a few plasma cells and mast cells, together with pigment accumulations, either free in the spaces or within cells, and granular debris of various kinds (Figs. 14 and 269). The subsequent fibrous thickening of the vessel wall in the advancing sclerosis has been traced by Lhermitte and Guccione to the transformation of plasma cells into fibro-blasts. In this investigation quite characteristic plasma cells were present in extremely few sections. Schob, Oppenheim, and Siemerling and Raecke found them in numerous instances, and look upon them as the expression of a more or less chronic process. Volsch and Flatau and Koelichen think most of the cells in late

stages belong to the lymphocytes, and that plasma cells are rarely found in the vessel walls or in the meninges.

(5) OTHER HISTORICAL FEATURES.

1. *Form, Symmetry, and Distribution.*

Rossolimo first drew attention to the dependence of the sclerosed areas upon the topographical distribution of the blood-vessels. As the causal agent spreads itself by the blood channel, or the lymph channel accompanying the vessels, it was asserted



that the areas assumed certain well-defined forms. This apparent dependence upon the vessel regions was seen specially clearly in small wedge-like sclerosis situated at the periphery of the spinal cord, in which there was always found a condensed or even obliterated vessel radiating from the pia. When the area in the white matter was separated from the periphery by a zone of normal fibres it was usually round or oval. These basal forms were in agreement with Kadyi's experimental observations on the arterial distribution in the cord, to which we have already alluded. The lateral vessel passed transversely into the cord substance and its first branches were given off transversely, but the transitional vessels ran upwards and downwards in the long axis of the cord. The accompanying figures clearly show that the area of distribution of the transverse branches is wedge-

shaped, and that of the perpendicular branches is an elongated oval. Such vessels were looked upon by Kadyi and Mager as end-arteries, but there is no doubt that there is a considerable amount of overlapping, and that, in consequence of this, the areas supplied by them are not sharply defined. The blood-supply, likewise, of the groups of ganglion cells is not from one individual branch of the commissural vessels, but from several. The sulco-commissural arteries form in the substance of the anterior horn a rich and intimate network which holds the cell groups enweaved in its meshes.

In the course of this study several small areas have been followed up, serially, throughout their whole extent, and I have come to the conviction that the changes appear within, but do not coincide with, the area of distribution of the arteries, and that it must be extremely difficult to determine the territory of an artery. In literature numerous statements are made to the effect that the position and the definition of the areas corresponded completely to the distribution of individual vessels, but neither in the white nor the grey matter could this be definitely traced.

In the cord the prevailing form of the area at the periphery was wedge-shaped to a certain degree, but in the smaller isolated areas, in which one, or at most two, small lateral vessels were present, the definition was irregular and the shape was ampulla-like, with the neck of the ampulla (Fig. 84) at the periphery of the cord, or bowl-shaped (Fig. 87). When a large part of the peripheral portion of the cord was affected, perhaps through fusion of contiguous lateral areas, the sclerosis frequently mapped out the triangular portion between anterior and posterior root entry zones and extended inwards to involve the grey matter—the base of this triangle being sub-pial (Fig. 76). The areas within the white matter of the lateral columns were frequently round or oval (Figs. 86, 88), and often occupied the region of the crossed pyramidal tracts. In the posterior columns the areas assume specially an elongated oval shape (Fig. 91), the long diameter of which is pointed sagittally. The centre line of this oval may be either the posterior median septum or the paramedian septum: in its extension the involvement of the columns is often uniform, but occasionally more on one side than another (Fig. 89). When the ventral portion of the posterior columns is affected by a small, isolated area, it has usually a more or less triangular form, with

the base to the posterior commissure (Fig. 90). In its extension such an area involves both posterior horns, and may pass backwards on both sides of the median septum—the apex gradually approaching the periphery, but often leaving symmetrical islets of normal tissue in the angles near the posterior root entry zones on either side.

Isolated areas may occur in the grey matter of the cord, especially in the anterior horns. Such areas may apparently involve one group of nerve cells (Fig. 73), or may, rarely, map out the whole of one horn as a demyelinated area extends before it into the white matter. The most frequent involvement, however, of the grey matter is a peri-central sclerosis, which may be very marked at almost every level of the cord. In its extension this may pass anteriorly or laterally into the grey matter, or directly anterior or posterior along both sides of the anterior and posterior fissures, or both or all combined, so that in its further extension it involves almost the whole transverse section of the cord. The symmetry of such involvement is often very marked (Fig. 77), and is never more clearly brought out than where isolated islets of myelinated fibres are left at corresponding marginal parts of an otherwise demyelinated transection. Similar marginal areas in the region of the cerebellar tracts are also frequently left after an apparently symmetrical involvement of the lateral columns and adjoining portions of the grey matter.

The longitudinal extension of areas within the white matter is very varied, but sometimes reaches over several segments. Their form is usually an elongated oval, or a series of elongated oval areas have seemed to join on to one another at their adjacent ends (Figs. 30, 31). In the frontal longitudinal section of an isolated area in the posterior columns, it is possible to recognise how the numerous vertical branches of a vessel system are all involved and not one primary vessel stem. Very numerous segments were cut in serial longitudinal sections, both in frontal and sagittal direction. In some of these only traces of myelinated fibres could be found along the margins throughout the whole segment; in others, the primary oval area was densely sclerosed and was surrounded by a broad, early transitional zone which above and below had a narrow wedge shape; and in others, as the area in the posterior columns was left behind, further serial sections showed the involvement of the posterior horns, the commissures and lateral tracts in one

extensive area. The innumerable variations can thus only be hinted at, but one further feature must be mentioned. Such long stretches—extending sometimes over two upper dorsal segments for instance—showed that definite areas in the same columns were united by faintly-staining tissue, which sometimes showed Marchi degeneration, or such an appearance was continued onwards, ascending or descending, from definite sclerotic tissue. The illustrations sufficiently indicate that the cervical cord and dorsal cord were more frequently and more extensively involved than the lumbar and sacral cords.

In the medulla oblongata, pons, cerebral peduncles and cerebellum, the same two primary forms were present with numerous variations. Sub-pial areas were frequently wedge-shaped, and these within the substance of the tissue round or oval, with the long diameter in frontal or sagittal section. Here, too, the confluence of the areas was marked. The cranial nuclei shared in the general extension from the floor of the IVth ventricle—an extension which usually took place in isolated broad processes which subsequently fused with one another. The extension of the sclerosis on the floor of the IVth ventricle passed laterally and, from the angles of the ventricle, inwards to involve the structures which at various levels are found in this region. The figures and the detailed topographical description sufficiently indicate the distribution. The lateral extension to the roof of the IVth ventricle involved the white matter of the cerebellum, the hilum of the dentate nucleus, the nuclei of the roof, and often the whole of the folia forming the vermis and nodules.

The peri-ventricular areas showed also numerous primary, wedge-shaped areas with broad base to the ventricle, and extensions into the adjoining tissue in the form of finger-like processes or ampullæ, in each of which a central vessel could usually be found. All these areas were often united by tissue staining faintly in Weigert sections. Around the posterior and anterior horns of the lateral ventricle the sclerosis assumed the form of a hood, the apex of which was continued in the direction of the frontal and occipital poles by a series of rounded or oval small areas.

In the medullary rays numerous submiliary foci were present, round or oval, with the long diameter parallel to the course of the fibres. The areas in the medullary rays were often united by

tissue in an earlier stage of degeneration. This gave a striped appearance even macroscopically. The peri-ventricular sclerosis of the descending horn of the lateral ventricle in almost every instance extended from the adjoining white matter in radiating lines into each medullary ray. In the transition zone between cortex and white matter the affected area often assumed the form of an elongated spindle, and when this zone lay in the cup of a convolution the spindle curved on itself, with the concavity to the fissure. In the extension of either flat or curved spindle, the poles often passed to involve the transition zone between two or more adjoining convolutions, and in this extension the whole of the white matter from which the individual medullary rays arose was often cut across.

In the large basal ganglia, isolated areas were often quite round (Fig. 96), but the fusion of primary areas in the optic thalamus, internal capsule, and lenticular nucleus produced large irregular areas in which might be traced the original forms. Symmetrical involvement of the basal ganglia and especially of the external capsule and claustrum, with an extension to the grey matter of the convolutions of the island of Reil, formed a prominent feature in several of the sections, especially in Case II. The distribution of the areas in the basal ganglia in individual cases has been given elsewhere, but here it may be noted that such areas seemed often to start in a peri-vascular zone around the lenticulo-striate and strio-thalamic vessels (Fig. 97), and that the peri-vascular sieve-like areas were nowhere so marked as in this region, around the larger of these branches.

In the cerebral cortex the variation in the form of the areas is even more marked than elsewhere. These may be grouped into three divisions according to whether the demyelination (1) spreads from white matter into grey and is arrested within the radiations of the nerve fibres, or (2) is wholly within the cortex with a zone of intact nerve fibres forming a fine network superficial to it, or (3) spreads from the surface and extends for a variable distance inwards. The shape of those extending from the white matter is usually dependent upon the subcortical portion, and the extension may be simply a gradual one which involves the radiations in a sinuous or curved or even pointed outline. The definition of the cortical portion within the radial fibres is often very sharp, even under a high power, but frequently individual fibres or even the

groups of fibres forming a single radiation may pass in from the white matter into the area for a considerable distance. In the areas, situated as far as could be made out by numerous serial sections, entirely within the cortex, no special shape could be noted. It was possible to trace small areas entirely within the tangential fibres (Fig. 130), or involving the fine fibres forming the supraradial network or cutting across the Baillarger's or Genari stripe, but more often these small isolated areas were found limited to the intra-radial and radiating fibres, which pass, with a fan-like arrangement, from the tips of the medullary rays. In many such areas a small vessel, which seemed to be the centre of the demyelination, was certainly found, but others bore no relation to any of the numerous capillary vessels found within them. The shape of the areas, passing in from the surface of the convolutions, was often that of a modified wedge, or of an arch with its convexity either on the surface or within the radiating fibres, and the outline of these areas which seemed to pass definitely over into the subcortical white matter was, as a rule, much sharper than that of those confined to the cortex (*cf.* Figs. 95-118).

The close disposition of the numerous areas in certain convolutions gave place often to a coalescence, with a complete demyelination of cortical tissue extending over the whole of one convolution and sometimes round the cups of the adjoining convolutions. This demyelination in some cases affected the cortex irregularly, and the resultant line might further be broken into by wedge-shaped or bow-shaped areas extending further inwards (Fig. 106). Short of complete coalescence of the areas, there were found stretches of the cortex which presented a moth-eaten appearance, in which only islets of the normal radiations or intra- or supraradial network of fibres were retained. Such a moth-eaten appearance was specially well brought out when the section was cut in a plane at right angles to the radiations (Fig. 125). It is perhaps necessary to emphasise that such descriptions are taken from specimens in which it could be stated almost definitely that the demyelination was not due to over-differentiation. The limits of such areas are quite defined; the adjoining convolutions have their tangential fibres and the fine fibres of the supraradial network well brought out; and the demyelination often corresponded exactly to the area of supply of the superficial vessel plexus of the cortex (*cf.* Figs. 101, 102),

although no single larger vessel could be brought into relation to it. Such an extensive affection of the cortex, too, is often limited to certain convolutions. Siemerling and Ræcke, Sanders, Schob, and others have pointed out similar extensive involvement of the cortex, and Alzheimer and Spielmeyer in general paralysis have noted the existence of demyelinated areas involving the cortex extensively, in which none of the cellular changes of general paralysis were present.

The remarkable symmetry of the cortical areas is again sufficiently indicated in Figs. 101-103, in one of which symmetrical areas on either side of an individual sulcus may be noted (Fig. 103).

In the cerebellum the cortex often shares in the extensive involvement of the cerebral cortex. Here the medullary cores of individual folia may be simply cut across, or the fine reticulum of nerve fibres in the granular layer may be also affected; at other times, especially in the flocculus, the trunk of numerous medullary cores is cut across, and this involvement spreads to affect individual branches and the corresponding reticulum of fibres, while immediately adjoining cores, with the pertaining cortical reticulum, are unaffected. A striking parallelism may be presented in the two flocculi (Figs. 27, 28)—the peduncles of which may also be involved in an extension from the angles of the IVth ventricle.

It is thus seen that the areas are apparently distributed over the whole central nervous system, that their form can be related only in a modified way to two basal types—wedge-shaped and round; and that a remarkable symmetry can be recognised in their localisation—a symmetry which can easily be underestimated when the areas are at different stages of development.

2. Secondary Degeneration.

The absence of secondary degeneration has been, till recently, accepted as one of the cardinal characteristics of the histological picture. Charcot related this feature to the preservation of the axis cylinders in the sclerotic tissue, and Schultze has shown that complete demyelination of the nerve fibre at a circumscribed level occasions no secondary degeneration even in the myelin sheath itself, and that this follows only a considerable alteration of the axis cylinder itself.

The question of secondary degeneration is one not easily decided. A glance over the numerous illustrations of the spinal cord at successive levels will suffice to show that the zones of sclerosis are not followed in the usual sense of the term by secondary degeneration. It is probably frequently present, however, especially in late stages, but the ordinary difficulties of proving or recognising it in cases where numerous islets of sclerosis are present are increased by numerous factors. We have already referred to the frequent presence of diffuse changes which unite sclerotic areas, or, on longitudinal section of the cord, are continued upwards or downwards as "Markschattenherde"; it is probable that some at least of these changes must be related to a commencing secondary degeneration. The destruction of the axis cylinders is, again, only relative in any one area, and in consequence, the secondary degeneration will affect only a certain percentage of the nerve fibres of one column or even of one bundle; its recognition as a secondary degeneration is thus rendered more difficult. Further, the sclerosis below or above any one level passes over the boundaries of a system degeneration, and may even affect the whole transverse section of the cord—the element in this change due to secondary degeneration would be impossible to trace. And, finally, more acute processes arising in the affected columns, without any loss of axis cylinders in areas above or below may be explained by an analogous process in the area under consideration. It may also be noted that numerous, though only microscopically evident, areas may frequently be found in the course of one and the same conducting tract.

Changes outside the areas, such as the diffuse alterations of the myelin sheath, diffuse commencing alterations of the glia cells, similar dilatation and engorgement of blood-vessels, and sieve-like dilatations of the adventitial lymph spaces and of the peri-vascular glia meshes, as the single expression of a not otherwise provable change, have all been referred to in the previous study. In late stages the blood-vessels throughout the whole central nervous system, and especially in the convolutions, showed varying slight changes, similar to those within the areas. Anton and Wohlwill have found the vessel infiltration sharply limited to the areas, but dilatation and engorgement throughout the whole cerebral white matter.

(6) CONCLUSION.

1. *General Features and Distribution of Areas in Case I.*

The number of areas was very large, and their distribution throughout the central nervous system was very extensive. The parts most involved were the cervical enlargement of the spinal cord, the medulla oblongata, the pons, and the peri-ventricular tissue. The most striking histological feature was that almost every area showed evidence of an advancing process (Figs. 64-69). The areas were thus wholly in an "early" stage or showed a peripheral advancing zone around a central condensed zone, and the impression the latter areas left was that the primary affection had never died down, but had gradually extended peripherally, while the central sclerosis had also extended eccentrically. A further striking feature was the presence of very extensive areas of "shadow" sclerosis, which united the areas of demyelinated tissue. It is unnecessary to enter into the structure of the areas, as this has been fully given in the earlier part of the study; the great majority were at the height of the fat granule formation and had not reached the stage of commencing glia fibril formation.

In the spinal cord the cervical enlargement was very markedly affected; the dorsal cord much less so, except at the level of D 10, which showed a complete transection (Fig. 58): the lumbar cord was also comparatively slightly affected, but almost the whole sacral cord showed a demyelination (Fig. 63). The most striking feature of the cord affection was that with one or two exceptions no isolated areas could be traced. The exceptions were in the dorsal cord, and one of these, cut in serial sections, extended about one-third centimetre in longitudinal extent, and at each end gradually passed into normally staining fibres. The central portions of the posterior and lateral columns were the most densely sclerosed, but round the periphery of even these parts there was a zone more or less wide of fat granule cells; and radiating vessels, with their lymphatic sheaths filled with similar cells, passed from this zone to the circumference of the cord. The frequent symmetry is well brought out in Figs. 49 and 59. Bielschowsky preparations showed a marked diminution in the number of axis cylinders, and those persisting were swollen and stained faintly. The ganglion cells showed all stages of degeneration, and at no level were they normal: numerous atrophic forms with central chroma-

tolysis and absence of processes could be found in the demyelinated tissue at almost every level, where the grey matter was affected (Fig. 240). The membranes of the lumbo-sacral cord were slightly thickened and infiltrated with cell elements.

The areas in the medulla oblongata and pons were much more sharply defined, as a rule, than those in the cord. Their most striking feature was the large number that showed a zone of "shadow" sclerosis around them. This was specially evident in the areas occurring amongst the transverse fibres of the pons and grey nuclei, and such shadow sclerosis had here the same defined outline as the central area. The nuclear areas on the floor of the IVth ventricle were markedly involved and many of the cells filled with a dark brown pigment. Nearly all the cranial nerve roots, on both sides, entered into demyelinated tissue. The areas have characters similar to those of the cord, only a few showing any definite central condensation. On the other hand, large irregular areas were found which showed a complete demyelination without any marked glia cell proliferation or blood-vessel change or alteration in the axis cylinders either in number or calibre. A very beautiful illustration of such an area is seen in Figs. 16, 17, and 251, in which can be seen the sharp, irregular outline of the demyelinated tissue and the continuation onwards of the axis cylinders, which intersect in the median raphé. These axis cylinders are tortuous and delicate, and their density, in Bielschowsky preparations, is the same as in normal conditions. The blood-vessels here are dilated and their walls also impregnated by the silver. The distribution of the areas in relation to the floor and walls of the IVth ventricle, and to the hilum of the dentate nucleus will later be referred to, but attention must here be drawn to the prominent involvement of numerous foliæ in the cerebellum and of the almost symmetrical affection of the flocculi (Figs. 27, 28) and of their peduncles.

The peri-ventricular sclerosis gave the impression of being the result of the fusion of sub-ependymal areas, and in individual sections such primary wedge-shaped areas, with base to the ventricle, could be recognised. Around the lateral ventricles and their horns, on all sides, large isolated areas could be traced in the white matter; their connection with the peri-ventricular sclerosis, especially in the areas above the roof of the lateral ventricles, could frequently be definitely proved. Numerous fat

granule cells were found distributed throughout the whole periventricular tissue, and in the walls of the sub-ependymal veins. The ventricles were not dilated, their walls were smooth, and there was no change in the ependymal epithelium.

The cortical areas were not very numerous compared to those found in one or two other cases, and there was an almost complete absence of the irregular demyelination of the superficial layers of the cortex. The contrast between the fat granule cells of the grey matter and those of the white matter was often well brought out (*cf.* Figs. 69 and 226). Bielschowsky preparations of the cortical areas showed numerous axis cylinders.

The optic nerves were extensively involved; the right was wholly demyelinated and showed a marked thickening of the connective tissue and glia trabeculae (Fig. 274). In the left nerve only one narrow strand of myelinated fibres could be found, but its whole length showed rows of fat granule cells (Fig. 65).

2. *Topographical Distribution in Weigert Sections.*

Spinal Cord.

Cervical Region (Figs. 48-54).—At the upper part of the cervical cord only a deficient staining of the myelin was found, involving the antero-lateral column of white matter with the adjacent anterior and lateral horns, but sparing the area occupied by the direct pyramidal tract. A few sections lower this area increased in size and extended mesialwards to the anterior median fissure. A second area, of more complete sclerosis, is now present, involving the columns of Goll along their anterior two-thirds. This area increases very rapidly and soon forms a broad band along each side of the mesial line, both anteriorly and posteriorly, involving grey and white matter indiscriminately. The symmetrical distribution of the sclerosis is here very marked. Slightly lower, the early "shadow" area has almost disappeared, the large mesial area now extends laterally to involve the greater part of the posterior horn and column on one side and the corresponding third of the other side, and a third area has appeared on the opposite side in the crossed pyramidal tract.

In the third segment four well-marked areas are visible; the largest involves the whole of the right lateral column with the exception of the fibres of Lissauer's tract; on the same side a small

triangular area extends outwards from the tip of the anterior horn. On the left side an irregular patch is present in the antero-lateral region, separated from the anterior horn by a narrow zone of normal fibres, while the fourth patch, roughly quadrilateral in outline, is found in the posterior columns and involves the centre of the posterior columns, extending slightly beyond the mesial line on each side. At a slightly lower level, each of these patches extends and leaves the normal tissue in the form of the letter H, while only a few sections lower there is almost a complete transection of the cord.

A section across the upper part of the cervical enlargement exhibits only a small area, along the periphery of the left antero-lateral region of the cord, where the fibres were unaffected, and even this area had a small patch of sclerosis about the middle. On the opposite side small isolated groups of normal fibres are present near the internal margin of the anterior horn and at the posterior root entry zone. In C6 the previous groups of uninvolvement have almost disappeared. On the right side, however, the direct pyramidal tract and the greater portion of the posterior columns are unaffected, and in C7 the normal tissue is increased—the greater part of the posterior columns on both sides being normal, as well as a portion of the right lateral and left anterior columns. In C8 the normal tissue is again much diminished, and consists of fibres on either side of the anterior median fissure and a portion of the posterior columns. At a slightly lower level the posterior columns become involved, only a few scattered fibres here and there escaping—the lateral column on one side, and the antero-lateral on the other also escape. In the lowest part of C8, the only lesion is one large patch on one side, limited by the posterior horn of grey matter, extending over the whole antero-lateral portion, but allowing the direct cerebellar tract to escape.

Dorsal Region (Figs. 55-58).—In D1 this patch of sclerosis has spread across to the opposite side, leaving only a small area on the lateral part unaffected. In the third segment only two early areas are found; one on the right side, involving the crossed pyramidal tract, the posterior horn, and the adjoining fibres of the column of Burdach; the other on the left side, extending lateralwards from the anterior horn. In the fifth segment there is one small oval patch in the region of the septo-marginal tract, and in the next segment two small patches appear, one in the centre of the left

crossed pyramidal tract and the other towards the outer part of the column of Burdach. Still lower the whole of the anterior and antero-lateral columns with the anterior horn are completely sclerosed, with the exception of a few fibres along the direct cerebellar tract. In the eighth segment the sclerosis, which forms a quadrilateral around the central canal, involves each anterior horn completely and the anterior fourth of the posterior columns. At the junction of D10 and D11 there is a complete transection of the cord, except a few fibres along the outer border of the anterior columns.

Lumbar Region (Figs. 59-60).—At the level of the second segment one lateral patch involves the crossed pyramidal and ascending cerebellar tracts; in L3 this area shows an early "shadow" sclerosis, and another patch, circular, around the central canal, extends forwards along both sides of the anterior median fissure. Here also the sclerotic tissue shows a marked symmetry, which is slightly masked by the shadow sclerosis in the lateral region. At the junction of L4 and L5 both these areas are normal, and three other isolated patches are present—one in the left antero-lateral column, one in the right lateral column, and one along the two sides of the posterior fissure.

Sacral Region (Figs. 61-63).—In the upper part one small patch passes forwards from the tip of the anterior horn to the periphery, and on the left side three smaller areas are present related to lateral vessels. At the third segment one large area involves almost the whole of one side of the cord, with the exception of a small portion near the median line. This area increases as we pass downwards, until it involves the whole of the lower sacral region and conus. The nerve roots of the cauda are normal.

Medulla Oblongata.

Just above the decussation of the pyramids one small patch is found at the tip of the right substantia gelatinosa Rolandi—a patch which increases in size as it is traced upwards, when a symmetrical area develops on the opposite side (Fig. 32). At the level of the middle of the inferior olive there is a large area in the interior of the left olive (Fig. 35), and a second smaller area at the margin of the opposite restiform body. Still higher, these patches become larger and more prominent, and involve almost the whole of one inferior olive and extend from it along the side of the medulla to the resti-

form body, which is also sclerosed. The opposite olive and restiform body also show small areas, and another small triangular area is present between these structures, in the position of the tract of Gowers.

At the upper level of the medulla (Fig. 38) both lateral margins are involved by irregularly-shaped patches. On the one side the outer half of the pyramid and of the inferior olive, together with the whole of the restiform body and the intermediate structures, are affected. On the opposite side the posterior outer quadrant of the inferior olive is involved in an area which sends a small prolongation into the *formatio reticularis*, and extends upwards to involve the outer parts of the restiform body—leaving the fibres in the middle intact and connected with the *formatio reticularis* by a narrow band of normal fibres. The entering VIIIth nerve on each side passes directly into sclerosed tissue; the fibres in their extramedullary course stain normally. The vermis and the accompanying nodules are also involved, and the sclerosed area extends on each side of the roof of the IVth ventricle, and passes into the hilum of the dentate nucleus—involving on one side the grey matter of the nucleus. Several of the medullary cores of the folia of the flocculus are demyelinated (Figs. 27, 28, 39).

Pons Varolii.

As we ascend the brain stem the sclerosis becomes still more marked; the structures lining the floor of the IVth ventricle are particularly affected, and in the lower part of the pons both lateral angles are profoundly altered (Fig. 39).

At the level of the lower third of the middle peduncles (Fig. 40) patches are found reaching from the ventricle to the surface. The sclerosed area involves nearly the whole of one side of the pons, and extends over the median raphé to affect all the fibres of the mesial fillet; on this side the middle two-thirds of the pyramid escape, together with a small tongue-like projection of normal fibres passing upwards into the inferior olive. The sclerosis here extends backwards along the floor of the ventricle and the lateral angle to the roof, and also laterally to involve the centre of the corresponding flocculus. On the opposite side a small area is present external to the inferior olive. The sclerosed tissue cutting across the middle peduncle is constricted in the middle, and thus is divided into two areas—one at the corner of the ventricle, and the other at the

zone of entry of the VIIIth nerve, extends into the white matter of the cerebellum and the corresponding flocculus. In the cerebellar white matter two small isolated patches are found, while in the folia several separate areas are present—one of which involves the vermis, two the junctions of the white matter and the folia, and others cut across the medullary cores of individual foliæ. The cranial nuclei in this region, which was cut serially, are all involved, the nuclei of the VIth nerve, the cochlear nuclei, and the nuclei of Bechterew and Deiters.

Slightly higher (Fig. 41) the only normal tissue is in the middle line, both middle peduncles being obliterated. The normal tissue is intersected by several small areas in different stages of sclerosis, and is separated from the IVth ventricle by a broad band. On the one side the sclerosis of the middle peduncle involves the whole of the restiform body, the emerging root-entry zone of the VIIIth nerve, and the associated nuclei, and extends round the angle of the ventricle to the roof. Laterally the sclerosis extends into the white matter of the cerebellum, the remaining white matter of which also shows numerous irregular patches in early stages. Several of the cores of the foliæ are also cut across by other small areas.

Middle of the Pons (Fig. 41).—At this level the one middle peduncle is still involved by a large irregular area, which extends from ventricle to surface and involves most of the pyramidal fibres, the transverse fibres, the trapezoid fibres, the superior olive and associated nuclei, together with the nuclei around the IVth ventricle. On the opposite side the sclerosis of the middle peduncle is not so complete; one patch extends inwards from the surface and affects the outer pyramidal fibres, but does not extend further than the inner border of the trapezium. The floor, angle, and roof of the ventricle are also involved, and on this side two further areas occur—one destroying the superior olive, the other the trapezoidal and middle peduncle fibres; while a central area involves the trapezium again and the adjoining portions of the formatio reticularis.

Upper Part of Middle Peduncles (Fig. 42).—Here the aqueduct of Sylvius is surrounded by a well-marked ring of sclerosis, which extends to the surface, allowing only a few fibres of the superior cerebellar peduncles on each side to escape. On the one side this area extends laterally into the middle peduncle, almost completely

obliterating it and sending a tongue-like projection into the fibres of the trapezium. On the other side the anterior third of the middle peduncle is affected, and the sclerosis extends postero-mesially and involves the grey matter of the pons, the corresponding fibres, and the middle of the pyramids.

Upper Pons (Fig. 43).—The ring round the aqueduct of Sylvius is now slightly altered; the greater part of one superior cerebellar peduncle escapes, while the anterior part of the opposite peduncle is also free. The sclerosis extends laterally, and is continuous on the one side with a large area involving the middle peduncle. Three further sclerosed areas are found; one in the middle line involving the pontine grey matter and fibres, and irregularly-shaped, almost symmetrical areas which extend postero-mesially from the lateral side of the pyramidal fibres on each side, and involve two-thirds of their fibres with the corresponding connections.

At a slightly higher level the central oval area increases in size, while the sclerosis round the aqueduct diminishes, and is localised to one side.

Junction of Pons and Mid-Brain (Fig. 44).

Three irregular areas here extend backwards from the surface of the pons. One, in the mesial line, reaches almost to the mesial fillet. This area cuts across, in irregularly-shaped, sharply-defined lines, the intersecting fibres of the raphé and the adjoining fibres on each side. The other areas, also irregular in outline, pass backwards on each side of the pyramidal bundles, which are also partly involved. A fourth patch extends forwards from the aqueduct of Sylvius, in the middle line, and involves the posterior longitudinal bundle and the cells in the adjoining grey matter.

At a slightly higher level (Fig. 45) the mesial patch is found to extend inwards only a short distance from the surface, while another patch, commencing close to it, passes round the periphery of the pons on one side. Numerous other patches are present on the transverse section: three small areas towards the middle line; one, which reaches forwards from the aqueduct of Sylvius and extends laterally to involve the posterior part of one superior cerebellar peduncle; and another small area at the anterior level of this peduncle.

Still higher (Fig. 46) sclerosed areas are present on the surface of the pons—on one side extending inwards to involve the mesial fillet. The sclerosis round the aqueduct of Sylvius involves it completely and sends a small projection forwards. In the centre of one pyramid a round “shadow” patch can be clearly identified, a second similar area is present in the middle line at the level of the mesial fillet, and a third at the lateral border of the mesial fillet.

Mid Brain (Fig. 47).

The aqueduct of Sylvius is now free, but an area occupies the mesial line in front of the commencing decussation of the superior cerebellar peduncles, and another is found laterally and slightly posterior. A triangular area of sclerosis in the middle line extends inwards from the anterior surface, and two smaller lateral areas are present, one on the surface and one slightly internal. These areas are all sharply marked off from the surrounding tissue, and bear no relation to any of the structures through which they pass.

Sub-thalamic Region.

In sections at this level an irregular patch is found in the anterior half of the mesial plane. The sclerosis extends outwards on each side into the red nucleus, and there are several small but well-defined areas in the ansa lenticularis on both sides. The aqueduct of Sylvius is surrounded by an oval patch, which extends outwards slightly beyond the grey matter.

Cerebral Hemispheres.

- (1) Horizontal sections through the cerebral hemispheres at the lower part of the basal ganglia (Figs. 23, 24).

Peri-ventricular Sclerosis.—The most prominent lesion is that found at the posterior cornua of the lateral ventricles. These are both surrounded by large irregular areas of sclerosis, which extend at several points through the white matter to reach the surface of the brain. This is well marked on the inner side of both posterior horns, where the sclerosis cuts across the tapetum, the inferior longitudinal bundle, and the splenium, and reaches the surface at the parieto-occipital fissure. On the right side this area is continuous with another which extends from the

angle of the ventricle to the foot of the calcarine fissure. Several small isolated areas are found in the splenium itself. The anterior cornua of the lateral ventricles also show small patches of sclerosis at their tip, both very small in size however. The remainder of the ventricular surface is unaffected on the left side, and on the right side three isolated patches are found: a small oval area close to its anterior surface, a narrow band in the middle line, and a larger oval patch on the surface opposite the splenium. These are all related to the ventricular surface of the optic thalamus.

Basal Ganglia.—A small oval area of sclerosis is present in the centre of the left optic thalamus, but the right, except for its ventricular surface, is unaffected. A number of minute areas occur in the anterior and the posterior limbs of both internal capsules, while a larger area is found on the posterior surface of the mesial border of the right putamen. The left putamen contains two early patches. One area, in the right claustrum extends to involve two of the convolutions of the island of Reil. In the left claustrum there are six small round or oval areas, each of which extends into the white matter on each side, and one reaches as far as the putamen.

Convolutions.—The left frontal lobe appears normal with the exception of one strongly-marked oval area in the medullary ray and grey matter of the anterior part of the frontal operculum. An equally well-marked area is found at the posterior part of the calcarine region—extending from white matter directly to the surface. In the right hemisphere small areas occur in the white matter of the frontal lobe; a slightly larger one is present towards its anterior margin, extending into the medullary ray and grey matter of the gyrus. Two minute early areas are present in the middle of the white matter of the frontal operculum, and a number of narrow, irregular areas—some extending for a distance of over a centimetre—are found limited to the cortical grey matter of this operculum, both on its outer and inner aspects, and on the surface of the island of Reil itself. Similar patches occur in the greater part of the parietal region, together with a large number of minute patches and one larger one at the junction of the white and grey matter in the cuneus. In the occipital lobe, a large number of minute patches, at all stages of development, are present both in the white and grey matter. These are grouped mostly towards the mesial surface and especially around the cal-

carine fissure. A few early areas occur also extending posteriorly from the peri-ventricular area already described

(2) At the level of the roof of the lateral ventricle (Fig. 26).

The most striking feature at this level is the large irregular area occupying the whole of the outer wall and posterior tip of the ventricle. This sclerosis extends irregularly into the adjacent white matter and completely cuts across the superior longitudinal fasciculus. A number of isolated areas, in different stages of development, are found along the mesial wall of the ventricle; these extend into the corpus callosum. Several large round and oval well-defined areas are found in the white matter, especially towards the occipital lobe—the largest of these measuring $1 \times \frac{3}{4}$ centimetre.

In the convolutions one small area is present at the tip of the frontal lobe; another occupies completely the medullary ray of the post-Rolandic gyrus; another is at the junction of the white and grey matter of the pre-cuneus, and two others, both circular in outline, occur in the grey matter of the calcarine region—one on the outer and one on the mesial surface.

The opposite surface of the cerebral hemisphere at this level shows a very similar affection and distribution.

(3) Above the ventricles (Fig. 25).

Numerous areas occur in the white and grey matter of both hemispheres. In the frontal lobe on the right side one large irregular patch occupies the centre of the white matter; several smaller areas are found on either side; and three early patches extend from the white matter into the medullary rays of the convolutions at the tip of the lobe. A small oval area is present in the post-Rolandic convolution at the junction of white and grey matter, while a smaller one is present in the white matter adjoining it. Another very well-marked area is present in the calcarine fissure; this extends from fissure to fissure, undermining completely the upper portion of the arcus parieto-occipitalis. Two or three small areas, confined to the grey matter, are also present in this region.

(4) Horizontal sections through the temporo-sphenoidal lobe (Fig. 29).

Sections just below the floor of the descending and posterior horns of the lateral ventricle show on both sides large irregular areas of sclerosis, which represent the downward continuation of the peri-ventricular sclerosis which has affected both these horns. The areas extend from the white matter almost to the surface of the convolutions, and the anterior margins of the descending horns are similarly affected. The sclerosis involves the greater part of the course of the occipito-temporal bundle of fibres; the convolutions in the region of the calcarine fissure are extensively involved—especially in the more superficial fibres of the grey matter; similar but more defined areas are present in almost all of the convolutions at the tip of the temporo-sphenoidal lobes; and the fibres of the hippocampal convolutions are also markedly affected.

3. Note on the Pathological Physiology.

There is no disease of the nervous system in which the symptoms may be more varied, in origin and in succession, than in disseminated sclerosis, a fact which can be well understood when one considers the wide range and irregular distribution of the areas of sclerosis. These, as we have seen, may occur in almost any part of the nervous system, producing peculiar symptoms and combinations of symptoms which may be of short duration, and are usually followed by periods of remission, after which a new series of symptoms may appear. Once definitely established, the disease is almost invariably fatal. The great variation in the nervous symptoms has resulted in a tendency, on the one hand, for any disease of nervous origin, of which the symptoms are unusual or difficult to interpret, to be classified as "disseminated sclerosis," while, on the other hand, the way in which the symptoms of a case of true disseminated sclerosis may simulate other nervous diseases has often led to errors in diagnosis. On account of the long duration of the disease in most cases, and the unsatisfactory results of treatment, patients are not kept in hospital for long periods of time, and thus the number of cases which come to autopsy is not large.

The earliest symptoms of the disease are extremely variable, and depend solely upon the particular part of the nervous system affected by a patch. There is, however, a tendency for these patches to occur more frequently in certain regions, and thus certain clinical symptoms appear more commonly than others, and

as a result are considered more or less pathognomonic of this condition.

Of these symptoms one of the commonest is weakness of the legs. This is met with in a very large number of cases, and is associated in most with involvement of the spinal cord. It usually is progressive, and a spastic paraplegia develops. It is often accompanied by a number of more or less well-defined sensory changes. Such symptoms may result from one or more patches in the cervical or dorsal regions of the cord, involving the descending motor and adjacent sensory fibres. If the area involved be still lower, *e.g.*, in the sacral region, then the only symptom may be a sphincter involvement. These spinal symptoms may be the only ones present, or they may be accompanied or followed by others due to involvement of the higher centres. Of these, the principal ones most commonly found are nystagmus, alteration in speech, and volitional tremor. These are largely due to want of co-ordination, and are the result of sclerosis spreading in from the ventricles.

The eye symptoms are of special importance, and are very often the first sign of the disease. They often disappear quickly or may ultimately proceed to total blindness. They are the result of patches in different places in the optic path, the optic radiations as they pass near the descending horn of the lateral ventricle being commonly involved in the peri-ventricular sclerosis. The later and more serious symptoms, however, result from involvement of the more peripheral part of the optic path, namely, the optic tracts and chiasma. The affection of the eye muscles occurs so frequently from the close relationship of the structures innervating them to the ventricular spaces, the peri-aqueductal sclerosis found in the mid-brain involving the third nucleus in many cases. The nucleus of Deiters and other vestibular nuclei situated at the angles of the IVth ventricle are also specially liable to involvement, and there is no doubt that it is the proximity of these structures to the ventricles which accounts for their frequent involvement, and the special diagnostic importance of nystagmus and visual symptoms.

The "volitional" tremor and scanning speech are also symptoms of the very greatest importance. They are both the result of defects of co-ordination. The tremor occurs only on voluntary movement and is absent during rest. In attempting to carry out

any movement the arm jerks about in an irregular manner, the tremor becoming quicker towards the end of the voluntary act. This form of involuntary movement, we know now, results from affection of the cerebello-rubro-thalamo-cortical path. Injury to this path allows involuntary movements to occur by removing the steadying influence which it normally exerts upon the Betz cells in the motor area. If this influence be removed, steady innervation of the anterior horn cells is impaired, and the more the pyramidal path is innervated, the more obvious does the tremor become. Involvement of the cerebello-rubro-thalamo-cortical path may occur thus at many different levels, and any of these lesions may result in this volitional tremor. That this path is specially liable to be affected is well seen in these cases here described, as in most of them patches were found at many different levels. They occur very frequently in the superior cerebellar peduncles, in the red nucleus, and in the optic thalamus.

The relation of the symptoms to the areas of sclerosis found after death is well seen in Case I. (L. W.). The symptoms in this case began with weakness of the legs, which passed off, recurred, grew rapidly worse, and terminated in a spastic paraplegia. This is obviously the clinical manifestation of the dense areas of sclerosis which were found throughout the spinal cord. These were obviously of long duration, and seem to have been the earliest manifestation of the disease. The patches which were found in the sacral region were also of an early date, and were responsible for the affection of the sphincters from which she suffered. The spinal cord lesions in this case were thus very extensive and appeared at an early stage of the disease.

The next symptom of importance to appear was the volitional tremor, with which we associate the patches in the superior cerebellar peduncle, red nucleus, and optic thalamus. These appeared also at a fairly early stage. The nystagmus was also an early symptom, and was undoubtedly associated with the peri-ventricular and peri-aqueductal sclerosis, only parts of the oculo-motor nucleus being involved.

After a short time the symptoms followed each other rapidly. An extension of the patches in the cervical region caused sudden numbness in the left arm. This was followed by a rapid development of large patches in the pons, one of which, involving the VIIIth nerve, produced deafness in the right ear, and another,

catching the facial nucleus, produced a right facial paralysis. These patches were found in an early stage and thus appeared late in the course of the disease. An extension still later of a patch involved the sixth nucleus, and led to diplopia from paralysis of the external rectus muscle. This was soon followed by the medullary patches which involved the XIIth nerve and nucleus, and led to a protrusion of the tongue to one side and slight difficulty in speech and swallowing. Later the vision suddenly became dim, and next morning she became totally blind. This was obviously the result of the development of patches in the optic tracts and chiasma. Later the areas in the spinal cord became still more extensive, and the effects of a lower neurone lesion became evident, resulting in muscular wasting and emaciation.

(Part V. will appear later).

DESCRIPTION OF PLATES.

(Figs. 1-22 are from drawings; Figs. 23-286 are micro-photographs).

The illustrations are grouped together to follow as closely as possible the different sections of the histological study. Plates IV-XVI give various levels of the brain and spinal cord in the different cases, and show the general topographical distribution of the areas (myelin sheath stain). In the remaining illustrations chief stress has been laid upon the evolution of the sclerotic areas and upon the sequence of the changes in the individual tissue elements.

PLATES I-VI :—

- Figs. 1-4. Evolution of a sclerotic area in the spinal cord through a stage of fat granule cell formation—longitudinal direction of the nerve fibres.
 „ 5-7. Ditto. Cerebral area: transverse direction of nerve fibres.
 „ 8-12. Ditto. Spinal cord: transverse direction of nerve fibres.
 „ 13-15. Sequence in blood-vessel changes.
 „ 16-17. Related to axis cylinders.
 „ 18-20. „ evolution of the fat granule cells (“Fettkörnchenzellen”).
 „ 21-22. Related to changes in the cerebral cortex.

PLATES VII-XII. Topographical distribution of areas in Case I.

PLATES XIII-XXII. Special features of spinal cord and cerebral areas. Weigert stain.

PLATES XXIII-XXVI. Special features of spinal cord and cerebral areas. Marchi method.

- FIGS. 155-172. Evolution of a sclerotic area—through a stage of fat granule cell formation: spinal cord—longitudinal direction of nerve fibres.
 „ 173-178. Ditto. Through a stage of increasing glia hyperplasia.
 „ 179-190. Ditto. Spinal cord—transverse direction of the nerve fibres.
 „ 191-199. Ditto. Cerebral white matter—transverse direction of the nerve fibres.
 „ 200-208. Ditto. Cerebral white matter—longitudinal direction of the nerve fibres.
 „ 209-214. Types of glia cell changes and glia fibril development.
 „ 215-226. Changes in cerebral cortical areas.
 „ 227-232. Positive and negative pictures—myelin sheath and neuroglia stains.
 „ 233-238. Changes in the transition zones of areas.
 „ 239-250. „ related to ganglion cells.
 „ 251-262. „ „ axis cylinders.
 „ 263-280. „ „ blood-vessels.
 „ 281-286. „ „ nerve roots, &c.

PLATE I.

Figs. 1-4. Successive stages in the evolution of a sclerotic area in the posterior columns of the cervical spinal cord. Sections are cut in the longitudinal direction of the nerve fibres (pp. 53-55), and show a gradually increasing glia fibril formation. (*cf.* figs. 155-166. Figs. 1 and 3, Ford-Robertson's methyl-violet stain; figs. 2 and 4, palladium methyl-violet. $\times 400$. *a*=glia nuclei; *b*=glia fibrils; *c*=fat granule cells; *d*=persistent axis cylinders.

PLATE II.

Fig. 5. An "early" area in the cerebral white matter (*cf.* fig. 191): shows a central blood-vessel (*b*), a peripheral nucleated zone (*d*), and is composed almost wholly of fat granule cells (*c*), and proliferated glia cells, seen only as nuclei (*a*) under this power. Heidenhain's iron-haematoxylin. $\times 26$.

Fig. 6. Area in the cerebral white matter. Numerous fat granule cells in the upper part of the drawing with an already advanced degree of fibril formation: few fat granule cells in the lower part with a still more advanced fibril formation—the glia nuclei almost wholly isolated from the fibril. *a*=protoplasmic glia cells with processes differentiated into fibrils; *b*=glia fibrils; *c*=fat granule cells; *d*=glia nuclei isolated from the fibrils. Heidenhain's iron-haematoxylin. $\times 350$. (Pp. 58-59). (*cf.* figs. 191-199).

Fig. 7. Types of proliferated glia cells with varying degrees of fibril formation. *a*=fibrils recurring near nucleus; *b*=glia nucleus forming nodal point from which fibrils radiate. (*cf.* figs. 212-214. Heidenhain's iron-haematoxylin. $\times 400$).

PLATE III.

Figs. 8-12. Successive stages in the evolution of a sclerotic area in the posterior columns of the cervical spinal cord. Sections cut transversely to the direction of the nerve fibres (pp. 55-56). (*cf.* figs. 179-190. Van Gieson's stain. $\times 350$. *a*=glia nuclei; *b*=blood-vessel; *c*=fat granule cell; *d*=myelinated nerve fibre; *e*=finely granular glia tissue; *f*=naked axis cylinder; *g*=transition to normal tissue.

Fig. 8. Shows a commencing enlargement of the nucleus, cell body, and processes of the glia cells and a commencing change in the myelin.

Fig. 9. Stage of glia cell proliferation and fat granule cell formation. Note the multi-nucleated glia cells, the presence of numerous deeply-stained nuclei in the tissue, the swollen and faintly-staining axis cylinders, and the "gitter" structure of the fat granule cells.

Fig. 10. Tissue is composed almost wholly of fat granule cells, many of which have accumulated within the adventitial sheath of the blood-vessels.

Fig. 11. Stage of advancing sclerosis: the glia fibrils, cut transversely, are represented as closely compressed fine dots; the persisting axis cylinders stain deeply; a few fat granule cells are still left in the tissue.

Fig. 12. Stage of advanced sclerosis. The tissue is dense and finally granular, contains numerous axis cylinders and a few fat granule cells—chiefly within the adventitial sheath of the capillaries. On the left transition to the normal tissue of the cord.

PLATE IV.

Figs. 13-15. Sequence of changes in the blood-vessels (pp. 119, 120). (*Cf.* figs. 263-280. Van Gieson's stain. $\times 350$. *a*=glia nuclei; *b*=blood-vessel; *c*=fat granule cell; *d*=cell containing blood pigment; *e*=lymphocyte-like cells; *f*=plasma-cell; *g*=glia tissue; *h*=connective-tissue cell.

Fig. 13. A transition vessel with its adventitial sheath filled with fat granule cells; numerous similar cells and enlarged glia cells in the surrounding tissue.

Fig. 14. A larger vessel (*cf.* fig. 269), during the stage of advancing sclerosis, to show the cell content of the adventitia after the removal of the fat granule cells.

Fig. 15. Small artery in an area of advanced sclerosis. Note the commencing "hyaline" fusion of the media and adventitia; the outer layers of the adventitia and the layers of the peri-vascular glia are still dilated and have an increased cell content. At a later stage the glia sclerosis immediately surrounds the dense, homogeneous vessel wall, which then shows few or no cell elements.

PLATE V.

Figs. 16-17. Persistence of axis cylinders across a demyelinated area in the pons. (*Cf.* figs. 119 and 258. Bielschowsky-Williamson silver impregnation method. Fig. 16, $\times 20$; fig. 17, $\times 90$. *a*=line of transition between myelinated and demyelinated fibres; *b*=median raphe where axis cylinders intersect; *x*=shown in fig. 17, under high power.

Figs. 18-20. Stages in the demyelination of an area and in the evolution of the fat granule cell. Frozen sections cut in the longitudinal direction of the nerve fibres. Scharlach R. $\times 300$. *a*=small glia nuclei; *c*=fat granule cell; *b*=transition forms between *a* and *b*; *d*=nerve fibre; *e*=blood-vessel; *f*=proliferated glia nuclei.

Fig. 18. Longitudinal nerve fibres immediately adjoining a demyelinated area. Note the cells containing fat granules.

Fig. 19. Structural elements in the transition zone of an early area. Note the longitudinal myelinated nerve fibres passing into the area, the rows of enlarged glia cells, and the numerous fat granule cells in all stages of their formation. The first fat granules are contained in the protoplasm adjoining the poles of the nucleus, and as they increase in amount the cell becomes round.

Fig. 20. Capillary, in a demyelinated area, completely surrounded by a layer of fat granule cells. Adventitial sheath not brought out in this stain.

PLATE VI.

Figs. 21-22. Glia changes in a completely demyelinated area in the cortex (pp. 93-98). *Cf.* figs. 131-226. Ford-Robertson's methyl-violet stain. $\times 500$. a = proliferated glia cells with protoplasm and processes differentiated into fibrils; b = capillaries with glia fibrils attached to their outer membrane; c = ganglion cells; d = small glia cells forming nests around the remains of ganglion cells; e = degenerated ganglion cells; f = retained axis cylinders. Note that the normal cyto-architecture of the tissue is preserved.

Fig. 21. Deepest (polymorphous) layer of the cortex.

Fig. 22. Layer of the deep pyramids.

PLATES VII-XII (CASE I.).

Figs. 23-63. Sections of brain and spinal cord. Kulschitzky-Pal myelin sheath stain with picro-fuchsin. Figs. 64-69. Sections to show the prevailing type of area present. Marchi method.

Figs. 23-24. Complete horizontal sections through the cerebral hemispheres at the lower part of the basal ganglia (pp. 141, 142). a = periventricular sclerosis around the posterior horns of the lateral ventricles, whence the sclerosis is continued backwards towards the calcarine fissure; b = the involvement of the splenium of the corpus callosum; c = area in the left optic thalamus; d = areas in the right and left claustrum extending to involve the convolutions of the island of Reil—small areas are present also in the anterior and posterior limbs of the internal capsules, and in the right and left putamen; e = oval area in the medullary ray and grey matter of the anterior part of the left frontal operculum (*cf.* fig. 114); f = two small areas in the right frontal operculum; g = several areas in the convolutions of the occipital lobe in both sides, especially around the calcarine fissure.

Fig. 26. Level of the roof of the right lateral ventricle. a = involvement of the outer wall and posterior tip of the ventricle; b = large areas in the adjoining white matter, especially towards the occipital lobe; c = area in the medullary ray of the post-Rolandic gyrus in the precuneus.

Fig. 25. Above the left ventricle—numerous areas in the white and grey matter especially. a = in the centre of the white matter towards the frontal lobe; b = at junction of white and grey matter in the post-Rolandic gyrus; c = area in convolutions of the calcarine fissure.

PLATE VIII.

Figs. 27 and 28, $\times 7$. Almost symmetrical involvement of the white matter of the flocculus of the cerebellum—an involvement (a) which extends into the cores of numerous foliae.

Fig. 29. Horizontal section through the temporo-sphenoidal lobe just below the floor of the descending horn of the lateral ventricle. a = extension

of the peri-ventricular sclerosis to the region of the calcarine fissure ; *b* = involvement of the convolutions at the tip of the lobe ; and *c* = of the fibres of the hippocampal lobe.

Figs. 30 and 31, $\times 2\frac{1}{2}$. Frontal longitudinal sections of the lower cervical cord from serial sections. In fig. 30 note the involvement of the anterior columns on either side of the anterior median fissure, with extension into the grey matter (*a*) and the lateral white matter (*b*). Fig. 31, through the central canal, with similar involvement of grey (*a*) and white (*b*) matter. Note the indication of the primary oval form of the areas.

PLATE IX.

Figs. 32-47 taken from Weigert serial sections of medulla oblongata and pons. $\times 2$. Note the distribution of the areas in relation to the floor and walls of the IVth ventricle ; the involvement of the dentate nucleus ; the frequent sharp definition of the areas ; that several are surrounded by a zone of shadow sclerosis, which has also a sharp outline ; and that nearly all the cranial roots enter into demyelinated tissue (pp. 137-141).

Fig. 32. Medulla oblongata above the decussation of the pyramids.

Fig. 33. At level of accessory olivary nucleus. *a* = peri-central sclerosis.

Fig. 34. At lower end of inferior olive.

Fig. 35. At middle of inferior olive.

Figs. 36 and 37. At the opening of the central canal into the IVth ventricle.

Fig. 38. Upper medulla continuous with the cerebellum. *a* = entering VIIIth nerve on each side ; *b* = demyelination of vermis and accompanying nodules ; *c* = involvement of the hilum of the dentate nucleus on both sides.

Fig. 39. Junction of medulla oblongata and pons. Note involvement of the fibres of the inferior and middle cerebellar peduncles on both sides, with extension to the centre of the corresponding flocculus. *a* = figs. 27 and 28 ; *b* = area in the cerebellar white matter.

Fig. 40. Pons Varolii at the level of the lower third of the middle cerebellar peduncle.

Fig. 41. Middle of pons. In figs. 38-41 note the involvement of all the cranial nuclei in this region—the nuclei of the VIth, the cochlear nuclei, and the nuclei of Bechterew and Deiters ; also the complete involvement of both middle cerebellar peduncles.

PLATE X.

Fig. 42. Pons at upper part of the middle peduncle. *a* = an area in the median raphe, which in higher sections (figs. 43-45) reaches the anterior surface.

Fig. 43. Upper pons. Note the symmetry of the involvement.

Fig. 44. Junction of pons and mid-brain. *a*=area in the middle line which reaches almost to the mesial fillet and cuts across, in sharply-defined lines, the intersecting fibres of the raphe and the adjoining fibres on each side. Cf. fig. 119 and figs. 16, 17, and 251, which show the complete retention of the axis cylinders across this demyelinated area.

Fig. 45. Slightly above (fifty serial sections) fig. 44. *a*=zone of "shadow" sclerosis around area.

Fig. 46. Fifty serial sections above fig. 45. Note three areas of "shadow" sclerosis. *a*=in centre of the pyramid; *b*=in middle line at level of mesial fillet; *c*=at lateral border of mesial fillet.

Fig. 47. Mid-brain. Aqueduct of Sylvius free. *a*=area in middle line anterior to the commencing decussation of the superior cerebellar peduncle.

PLATE XI.

Figs. 48-63. Various levels of the spinal cord. Myelin sheath stain, $\times 2$. (Pp. 135-137). Note that there are few isolated areas, and that there is frequent almost complete symmetry of the involvement (cf. figs. 49 and 59). Note further the complete demyelination at the lower dorsal (fig. 58) and lower sacral segments (fig. 63), and the complete myelination of the nerve roots of the cauda equina (figs. 62 and 63; cf. figs. 282 and 283).

PLATE XII.

Figs. 64-69. Marchi sections to show the prevailing type of areas present in optic tract and optic nerve (figs. 64 and 65, $\times 10$); in spinal cord (figs. 66 and 67, $\times 6$), and in the brain (fig. 68, $\times 75$ and fig. 69, $\times 10$).

Figs. 64 and 65. Showing "early" degeneration with rows of fat granule cells.

Fig. 66. Small "early" area in the posterior columns (cf. fig. 143).

Fig. 67. Posterior columns show a late stage of sclerosis with dense glia tissue staining darkly; both lateral columns show an "early" stage with rows of fat granule cells around the vessels.

Fig. 68. Typical small "early" area in the central white matter. The tissue around a central blood-vessel is permeated with fat granule cells.

Fig. 69. Demyelinated "early" area at the tip of a medullary ray, extending through the radiations almost to the superficial cortex—its medullary portion and the vessels radiating from it are permeated with fat granule cells.

PLATE XIII.

Figs. 70-79. Transverse sections of the cord from various cases to illustrate special features of individual areas. Kulschitzky-Pal with picro-fuchsin. Figs. 70-73, $\times 10$; figs. 74-79, $\times 6$.

Fig. 70. Sixth cervical segment. Note complete preservation of nerve roots.

Fig. 71. Fifth lumbar segment. Note lateral vessels (α) passing to the postero-lateral projection of the anterior horn (*cf.* fig. 83).

Fig. 72. Fifth lumbar segment. α =involvement of the glious, extra-medullary portion of posterior nerve root.

Fig. 73. Third lumbar segment at a low level. Note areas at the tip of the anterior fissure and around posterior median fissure. Also α =pericentral sclerosis, and b =area in lateral part of anterior grey matter.

PLATE XIV.

Fig. 74. Third cervical segment. Note the tendency towards symmetry and the varying stages of the involvement.

Fig. 75. Junction of seventh and eighth cervical segments. α =small isolated areas.

Fig. 76. Eighth cervical segment near the junction with first dorsal. Note α =large triangular area with base to surface of the cord—extension to the grey matter which is still outlined.

Fig. 77. Third lumbar segment. α =symmetry of involvement of the tissue around central canal and adjoining anterior median fissure.

Fig. 78. Fourth lumbar segment—incomplete symmetry.

Fig. 79. First sacral segment. Large irregular area with distinct outline.

Fig. 80, $\times 10$. Frontal longitudinal section of the cord showing complete demyelination. α =normal nerve roots; b =longitudinal small vessels with condensed walls.

Figs. 81-82. Upper and lower levels of first dorsal segment—from serial sections. Involvement simulates secondary degeneration.

PLATE XV.

Figs. 83-94. Special features of spinal cord areas. Kulschitzky-Pal with picro-fuchsin. Figs. 83-88 and 90-93, $\times 30$; fig. 89, $\times 40$; fig. 94, $\times 80$.

Fig. 83 (*cf.* fig. 71 (α)). Note α =lateral vessels passing to area which has picked out the postero-lateral group of anterior horn cells; b =wide glia marginal zone.

Fig. 84. Small oval area around an artero-lateral vessel.

Fig. 85. Larger area extending from the anterior surface of the cord to involve the anterior margin of grey matter (α).

Fig. 86. α =small oval area at junction of white and grey matter, around the terminal branches of a lateral vessel; b =smaller area near the surface around a lateral vessel.

Fig. 87. Wedge-shaped area with truncated apex.

Fig. 88. Small undefined area within the lateral column of white matter.

PLATE XVI.

Fig. 89. Triangular area in posterior columns with apex near posterior commissure (*a*). This area extends posteriorly to the surface of the cord.

Figs. 90-92. Indistinctly outlined, small areas around the anterior, middle, and posterior thirds, respectively, of the posterior median fissure.

Fig. 93. (*cf.* fig. 72 (*a*)). Demyelinated glious area in the immediately extra-medullary portion (*a*) of the posterior root and continuous with large area in the postero-lateral region of the cord (L5).

Fig. 94. Similar demyelinated glious area (*a*) immediately external to the "Ablassung" zone (*b*); intramedullary fibres normal (*c*).

PLATE XVII.

Figs. 95-106. Special features of cerebral areas: figs. 95-98, in the basal ganglia; figs. 99-106, chiefly involving the cortex. Kulschitzky-Pal with picro-fuchsin.

Fig. 95, $\times 5$. Two areas (*a*) involving both internal capsule and globus pallidus; a third area (*b*) cutting across the internal medullary lamina of the lenticular nucleus and extending into both globus pallidus and putamen.

Fig. 96, $\times 13$. Area around blood-vessel in the lenticular nucleus.

Fig. 97, $\times 6$. Areas, around vessels, involving *a*=optic thalamus; *b*=internal capsule; and *c*=lenticular nucleus.

Fig. 98, $\times 7$. Area in the white matter of the occipital lobe in the path of the optic radiations.

Fig. 99, $\times 6$. Convolutions around the calcarine fissure, showing involvement of the optic radiations (*a*) and of the cortical grey matter (*b*).

Fig. 100, $\times 6$. Convolutions around the opposite calcarine fissure of the same case as fig. 99, showing a large number of areas. *a*=small area confined to a medullary ray; *b*=areas involving both medullary ray and radiations; *c*=confined to the deep cortex; *d*=extensive demyelination of the superficial cortex.

PLATE XVIII.

Fig. 101, $\times 7$. Convolutions at the extremity of the occipital lobe; the medullary ray and radiations at the tip of both convolutions (*a*) all sharply cut off in an irregular line.

Fig. 102, $\times 7$. Convolutions at the extremity of the opposite occipital lobe of the same case as previous figure. *a*=complete demyelination of the superficial cortex over an extensive surface; and *b*=invasion of the deep cortex.

Fig. 103, $\times 7$. Superior frontal convolution. *a*=cutting off of the medullary radiations at the adjoining tips of two convolutions.

Fig. 104, $\times 7$. Convolutions surrounding the central fissure at the extreme vertex of the hemisphere. α =irregular demyelination of the superficial and invasion of the deep cortex.

Fig. 105, $\times 7$. Convolutions of the para-central lobule. α =area confined to the medullary radiations; b =wedge-shaped area involving whole depth of the cortex, and extending with truncated apex into the transition zone of white matter.

Fig. 106, $\times 6$. Convolution of the island of Reil. α =irregular demyelination of the cortical radiations and extension into the cups of the adjoining convolutions; b =small area in the white matter at base of the medullary ray.

PLATE XIX.

Figs. 107-118. Special features of cerebral areas, chiefly cortical. Kulschitzky-Pal with picro-fuchsin.

Fig. 107, $\times 6$. Convolution of the para-central lobule. Note the sharp delimitation of the area both in the white matter and in the radiations of the cortex (*cf.* fig. 74 (α)).

Fig. 108, $\times 15$. Convolution of the intra-parietal sulcus. Wedge-shaped area in the cortex extending with truncated apex into the white matter.

Fig. 109, $\times 13$. Convolution of the marginal gyrus. Area surrounded by a zone of lighter staining both in the white matter and cortex.

Fig. 110, $\times 12$. Convolution of the parieto-occipital fissure.

Fig. 111, $\times 10$. Superior parietal convolution. Involvement of the cup of a convolution.

Fig. 112, $\times 7$. Superior frontal convolution. Area extending from the medullary ray and sharply cutting off the radiations.

PLATE XX.

Fig. 113, $\times 5$. Convolution of the para-central lobule. Two well-defined areas in the white matter and one extending from medullary ray to surface of the convolution.

Fig. 114, $\times 6$. Convolution of the frontal operculum. Area cutting across the medullary ray and involving cortex on either side (*cf.* fig. 25 (e)).

Fig. 115, $\times 5$. Area in a parietal convolution involving several medullary rays with their radiations, with the exception of the tip of one convolution (*cf.* fig. 95 (e)).

Fig. 116, $\times 28$. Area at the lateral surface of a convolution—involving both transition zone and radiations, and showing the very abundant capillary plexus of the cortex.

Fig. 117, $\times 33$. Small area situated within the radiations, with central longitudinal vessel.

Fig. 118, $\times 13$. Convolution of the central fissure—well-defined area in the medullary ray and involving the transition zone (*cf.* fig. 74 (*b*)).

PLATE XXI.

Figs. 119-130. Special features of individual areas, chiefly cerebral. Kulschitzky-Pal with picro-fuchsin. Fig. 121, Heidenhain's iron-haematoxylin stain.

Fig. 119, $\times 10$. Area in the mesial line of upper pons—cutting across, in sharply-defined lines, the intersecting fibres of the raphe and the adjoining fibres on each side. *Cf.* fig. 44 (*a*); also figs. 16, 17, and 251, which show the complete retention of the axis cylinders across this demyelinated area.

Fig. 120, $\times 6$. Showing *a*=involvement of the medullary cores of several cerebellar foliae; *b*=areas in the cerebellar white matter; and *c*=in the peduncles.

Fig. 121, $\times 10$. Medulla oblongata at level of middle of inferior olive, showing faint staining of the pyramidal tracts. Section is given to illustrate the use of Heidenhain's iron-haematoxylin stain to bring out myelinated nerve fibres.

Figs. 122-124. Areas with central blood-vessels; figs. 122, $\times 30$, in external capsule and claustrum; fig. 123, $\times 30$, in central white matter; fig. 124, $\times 13$, in central white matter.

PLATE XXII.

Fig. 125, $\times 28$. Multiple, minute, demyelinated area in the superficial cortex: sections cut at right angles to the radiating fibres of the cortex.

Fig. 126, $\times 50$. Radiating fibres passing for an irregular distance into an area.

Fig. 127, $\times 10$. Bowl-shaped area in the superficial cortex.

Fig. 128, $\times 30$. Demyelinated area (*a*) showing no change in the cyto-architecture of the cortex (*cf.* next figure).

Fig. 129, $\times 50$. Demyelinated area (*a*) showing marked cell reaction in the cortex (*cf.* previous figure).

Fig. 130, $\times 30$. Slight demyelination (*a*) of the tangential fibres of the cortex.

PLATE XXIII

Figs. 131-142. Special features of early cerebral areas, in which numerous fat granule cells are present, both scattered in the tissue and collected around the blood-vessels. Marchi method.

Fig. 131, $\times 20$. Area in the central white matter.

Fig. 132, $\times 20$. Area confined to a medullary ray.

Fig. 133, $\times 20$. Area involving apex of a medullary ray and passing into the radiations.

Fig. 134, $\times 20$. Similar area with central (older) portion clearing up. Note that the longitudinally-running vessels, passing from the area, are outlined by fat granule cells.

Fig. 135, $\times 20$. Narrow area extending along the transition zone of a medullary ray.

Fig. 136, $\times 30$. Area distinctly limited to the medullary ray in one convolution, but passing in another into the radiations.

PLATE XXIV.

Fig. 137, $\times 20$. Involvement of the genu of the corpus callosum.

Fig. 138, $\times 30$. Peri-ventricular area around the descending horn of the lateral ventricle.

Fig. 139, $\times 20$. Involvement of the hilum and lamellæ of the dentate nucleus of the cerebellum.

Fig. 140, $\times 33$. Area in central white matter, showing clearing up of the central zone.

Fig. 141, $\times 20$. Area on the path of the optic radiations (*cf.* fig. 98). Note that the fat granule cells are confined to the peripheral zone.

Fig. 142, $\times 50$. Area, almost completely sclerosed, in which a few fat granule cells are present in both central and peripheral zones.

PLATE XXV.

Figs. 143-154. Special features of areas in the spinal cord : figs. 143-145, transverse section ; figs. 146-153, longitudinal section. Marchi method.

Fig. 143, $\times 35$. Small area around the vessels of the posterior median fissure (*cf.* fig. 66) ; fat granule cells, stained black with the osmic acid, permeate the tissue, and surround the capillary and larger vessels.

Fig. 144, $\times 100$. H.P. of previous figure.

Fig. 145, $\times 30$. Margin of cord with pia, showing the lateral vessels, with fat granule cells in their adventitial lymphatic sheaths, passing towards the inner layers of the pia, within which they spread in all directions.

Fig. 146, $\times 5$. Longitudinal interrupted lines of fat granule cells.

Fig. 147, $\times 7$. Ditto. Paler appearance of part of the section is due to the removal of the fat granule cells in the adventitial lymphatics.

Fig. 148, $\times 35$. H.P. of previous section, showing these cells in longitudinal rows.

PLATE XXVI.

Figs. 149-153. Evolution of the Marchi changes, in the nerve fibre, which lead to the formation of the fat granule cells ; early change frequently a

darkening of the myelin (fig. 149, $\times 50$); early degeneration in the form of rows of small globules (figs. 150-152, $\times 30$); gradual appearance of fat granule cells (fig. 153, $\times 200$).

Fig. 154, $\times 40$. Degeneration in the sciatic nerve in Case I.

PLATE XXVII.

Figs. 155-163. Evolution of an actual sclerotic area, in the posterior columns of the spinal cord, through a stage of fat granule cell formation (fig. 163); sections cut in the longitudinal direction of the nerve fibres (pp. 79-82). *Cf.* figs. 1-4. Figs. 155-161, $\times 200$, Heidenhain's iron-haematoxylin; figs. 162-163, $\times 200$, Palladium methyl-violet. *a*=glia nuclei; *b*=glia fibrils; *c*=fat granule cells; *d*=persistent axis cylinders; *e*=blood-vessels.

Fig. 155. Commencing reaction of all the tissue components.

Fig. 156. Fat granule cell formation with commencing glia fibril formation.

Figs. 157-158. Glia cell proliferation with glia fibril formation at the expense of the glia cell protoplasm and protoplasmic processes.

Figs. 159-160. Increasing glia fibril formation with gradual removal of fat granule cells.

PLATE XXVIII.

Figs. 161-163. Advancing and complete sclerosis.

Figs. 164-166. Variations in the final glia picture. Fig. 164, retained axis cylinders (darker lines) surrounded by parallel coursing fine glia fibrils. Methyl-violet, $\times 200$. Fig. 165, undulating lines of glia fibrils and thickened longitudinal vessels. Kulschitzky-Pal and picro-fuchsin, $\times 50$. Fig. 166, sclerosed area with numerous glia nuclei haematoxylin and eosine. $\times 200$.

PLATE XXIX.

Figs. 167-172. Low-power view of the evolution of an actual sclerotic area through stages similar to those in previous plate. Longitudinal sections of the posterior columns of the spinal cord. Van Gieson's stain, $\times 70$. Fig. 171, $\times 50$. Letters *a-e*, as in previous plate; *f*=still myelinated nerve fibres; *g*=dense sclerotic tissue.

Fig. 167. Commencing glia proliferation and fat granule cell formation. Note the rows of large protoplasmic glia cells (fig. 209).

Fig. 168. Typical picture of "early" area in stage of so-called "fat granule cell myelitis."

Fig. 169. Increasing glia fibril formation.

Fig. 170. Gradual condensation and removal of fat granule cells.

Fig. 171. Advanced sclerosis with their complete removal.

Fig. 172. Complete sclerosis; tissue consists of longitudinally coursing glia fibrils, blood-vessels, and a few persistent axis cylinders.

PLATE XXX.

Figs. 173-178. Evolution of an actual sclerotic area in the spinal cord through stages of increasing glia hyperplasia. Transverse sections of the lateral columns. Note, no fat granule cells are seen in any of these sections. *a*=glia nuclei; *b*=glia trabeculae; *c*=glia reticulum; *d*=naked axis cylinders; *e*=blood-vessel; *f*=myelinated nerve fibres.

Fig. 173, $\times 80$, and fig. 176, $\times 500$. Commencing thickening of the glia trabeculae and of the fine glia reticulum. Van Gieson's stain.

Fig. 174, $\times 50$, and fig. 177, $\times 150$. Gradual condensation of this reticulum. Van Gieson's stain.

Fig. 175, $\times 150$. Condensation and almost fusion of the glia reticulum. Note the still preserved axis cylinders and the enlarged glia cells. Cajal's silver method.

Fig. 178, $\times 200$. Shows lesser and more advanced degrees of the increasing glia hyperplasia. *tz*=transition zone. Cajal's silver method.

PLATE XXXI.

Figs. 179-184. Evolution of an actual sclerotic area (fig. 184) in the posterior columns of the spinal cord, through a stage of fat granule cell formation; sections cut transversely to the direction of the nerve fibres (pp. 71-79). Cf. figs. 8-11. Heidenhain's iron-haematoxylin stain. Figs. 176-181, $\times 370$; figs. 182-184, $\times 200$. *a*=glia cells; *b*=glia fibrils; *c*=fat granule cells; *d*=axis cylinders; *e*="Kielstreifen"; *f*=blood-vessels surrounded by layers of fat granule cells; *g*=central canal; *h*=dense glia tissue; *i*=glia fibrils forming whorls.

Fig. 179. Stage of glia cell proliferation and fat granule cell formation.

Fig. 180. Stage of so-called "fat granule cell myelitis."

Fig. 181. Advancing glia fibril formation.

Fig. 182. Fat granule cells collected in the adventitial sheaths of the vessels and gradually being drained away from the area.

Fig. 183. Stage of advanced sclerosis; no fat granule cells but finely granular glia and retained axis cylinders.

PLATE XXXII.

Figs. 185-186. Same evolution under low power. Small area in anterior third of posterior columns. Fig. 185, stage of advanced glia fibril formation. Van Gieson's stain. $\times 70$. Fig. 186, stage of advanced sclerosis. Van Gieson's stain. $\times 50$.

Fig. 187. H.P. of transition zone (*t*) of area similar to that in previous figure (*cf.* fig. 12).

Figs. 188-190. Variations in the final glia picture of the above evolution ; transverse sections of the posterior columns of the cord.

Fig. 188. Dense sclerotic tissue containing a few fat granule cells and numerous enlarged glia cells. Van Gieson's stain. $\times 200$.

Fig. 189. Showing glia whorls and irregular glia fibril formation. Van Gieson's stain. $\times 50$.

Fig. 190. Central "Kielstreifen," with numerous large glia cells and swollen axis cylinders in the dense sclerotic tissue on either side.

PLATE XXXIII.

Figs. 191-196. Evolution of a sclerotic area (fig. 194) in the cerebral white matter, through a stage of fat granule cell formation ; nerve fibres cut mostly transversely (pp. 82-80). *Cf.* figs. 5 and 6. *a*=glia cells ; *b*=glia fibrils ; *c*=fat granule cells ; *d*=axis cylinders ; *e*=blood-vessels. Heidenhain's iron-haematoxylin stain.

Fig. 191, $\times 40$; *cf.* fig. 5. Small "early" area with *e*=central blood-vessel ; *t*=transitional nucleated zone.

Fig. 192, $\times 60$. Stage of commencing glia fibril formation and fat granule cell formation ; *f*=fig. 195.

Fig. 193, $\times 60$. Stage of advancing glia fibril formation ; *f*=fig. 196.

Fig. 194, $\times 60$. Stage of complete sclerosis—a dense tissue with very fine meshes.

Fig. 195, $\times 300$. H.P. of fig. 192 (*f*).

Fig. 196, $\times 300$. H.P. of fig. 193 (*f*).

PLATE XXXIV.

Figs. 197-199. Variations in the density of the final glia network. Iron-haematoxylin. Fig. 197, $\times 150$, open network with a few persistent axis cylinders ; fig. 198, $\times 150$, denser network, especially around the capillaries ; fig. 199, $\times 80$, numerous glia nuclei which form the nodal points from which radiate glia fibrils.

Figs. 200-202. Evolution of a sclerotic area (fig. 202) in the cerebral white matter (medullary ray) ; nerve fibres cut longitudinally (p. 86). Heidenhain's iron-haematoxylin. $\times 200$. Note the persistence of the axis cylinders as swollen, homogeneous lines.

PLATE XXXV.

Figs. 203-208. Sclerotic areas in special situations. Iron-haematoxylin. Fig. 203, $\times 70$, in the middle cerebellar peduncle ; fig. 204, $\times 150$, ditto, showing a more advanced glia fibril formation ; fig. 205, $\times 200$, in the hilum

of the dentate nucleus ; fig. 206, $\times 38$, "early" peri-ventricular area ; fig. 207, $\times 200$, H.P. of previous figure ; fig. 208, $\times 150$, area cutting across a medullary core of a cerebellar folia.

PLATE XXXVI.

Figs. 209-214. Types of glia cell changes ; *cf.* figs. 221-226. Iron-haematoxylin. Fig. 209, $\times 500$, in the posterior column of the spinal cord ; rows of large, frequently multi-nucleated, protoplasmic glia cells ; fig. 210, $\times 600$, in the cerebral white matter ; protoplasmic potential fibril-forming cells ; fig. 211, $\times 200$, in the cortex ; nests of small glia cells (*a*) surrounding the ghosts of ganglion cells ; also (*b*) protoplasmic glia cells.

Figs. 212-214. Evolution of glia fibrils from large protoplasmic glia cells. $\times 600$. Note the definition of the borders of the protoplasmic processes (fig. 212), which can be followed throughout the concave border of two adjoining processes (fig. 213), and that the general outline of the fibrils corresponds at first to the general outline of the borders of these processes (fig. 214). Such cells are found very abundantly in the cerebral white matter and in the deepest layer of the cortex.

PLATE XXXVII.

Figs. 215-226. Changes in cortical areas.

Fig. 215, $\times 60$. Demyelination of cortex without any change in the cyto-architecture.

Fig. 216, $\times 45$. Demyelination of cortex with marked glia cell reaction in the transition zone and in the deep layers of the cortex.

Fig. 217, $\times 60$. Ditto. Note the marked alteration in the Betz cells (*a*). Figs. 215-217, Van Gieson's stain.

Fig. 218, $\times 80$. Cortical and subcortical area with glia cell reaction in the deep layers of the cortex.

Fig. 219, $\times 200$. Transition zone. *a*=glia cells ; *c*=fat granule cells.

Fig. 220, $\times 200$. Transition zone ; stage of advancing sclerosis. *a*=glia nuclei ; *b*=glia reticulum.

PLATE XXXVIII.

Figs. 221-226. Glia cell changes in the respective layers of the cortex ; *cf.* figs. 21 and 22. *a*=proliferated glia cells with numerous fibrils ; *b*=nests of small glia cells, around the ghosts of ganglion cells ; *c*=ganglion cells ; *d*=persistent axis cylinders ; *e*=blood-vessels. Note relation of the glia cell processes and fibrils to the capillary walls.

Fig. 221, $\times 200$, and fig. 224, $\times 600$. In the polymorphous (deepest) layer. Methyl-violet.

Fig. 222, $\times 200$, and fig. 225, $\times 600$. In the layer of the deep pyramids. Methyl-violet.

Fig. 223, $\times 200$. In the granular layer; nests of small glia cells around ganglion cells or replacing them. Hæmatoxylin and eosin.

Fig. 224, $\times 400$. In the layer of the large pyramids. Note ganglion cells (*c*) surrounded by enlarging satellite cells (*f*), whose protoplasm is filled with black-stained granules. Marchi method. Similar cells are found around the capillary vessels in this layer.

PLATE XXXIX.

Figs. 227-272. Low-power view of areas in myelin sheath and glia stains to show comparative negative and positive pictures.

Fig. 227, $\times 40$, and fig. 230, $\times 30$. Areas in the central white matter showing absence of myelin.

Fig. 228, $\times 60$, and fig. 231, $\times 30$. Similar areas to show presence of glia in the demyelinated tissue.

Fig. 229, $\times 50$. Very minute area in the central white matter showing slight demyelination.

Fig. 232, $\times 200$. Similar minute area to show the commencing enlargement of the glia cells (*a*) in this area of slight demyelination.

PLATE XL

Figs. 233-235. Transition peripheral zones in areas in the central white matter to show the glia nuclear proliferation. Van Gieson's stain. Fig. 233, $\times 40$, an old area with zone of small glia cells (*b*); fig. 234, $\times 200$, sclerosis still incomplete with large (*a*) and small (*b*) glia cells; fig. 235, $\times 60$, wedge-shaped zone of small glia cells (*b*).

Figs. 236-238. Transition zones of advancing areas in the spinal cord to show the mode of degeneration of the myelin. Frozen sections; Heidenhain's iron-hæmatoxylin. Fig. 236, $\times 60$, longitudinal myelinated fibres passing into an "early" area; fig. 237, $\times 300$, similar fibres under H.P. to show the fine globules and droplets of myelin which take the hæmatoxylin stain; fig. 238, $\times 300$, similar nerve fibres in transverse section.

PLATE XLI.

Figs. 239-249. Ganglion cell changes in the anterior horn of the spinal cord (except fig. 248) (pp. 110-112). Figs. 239-243, changes probably not related to the sclerotic process, but to the accompanying want of function, decubitus, &c.; figs. 244-249, changes probably related to both processes.

Fig. 239, $\times 40$. Ganglia cells very atrophic, but rounded forms present with nucleus and chromophile granules almost normal in structure and arrangement. Figs. 239-241, polychrome methylene blue.

Fig. 240, $\times 80$. Similar cells occurring in a demyelinated area.

Fig. 241, $\times 200$. Cells in a demyelinated area showing chromatolysis and excentric nuclei.

Figs. 242-243, $\times 75$. Cells showing marked pigmentation both in myelinated and demyelinated tissue. Weigert's myelin sheath stain.

Fig. 244, $\times 275$. Commencing reaction of the glia cells around a ganglion cell. Figs. 244-246, polychrome methylene blue.

PLATE XLII.

Fig. 245, $\times 200$. Atrophy and disappearance of ganglion cells with marked neuroglia cell reaction.

Fig. 246, $\times 200$. Similar to previous figure—occurring in the opposite anterior horn.

Fig. 247, $\times 200$. Ganglion cell, retaining its processes and chromophile structure, in the midst of sclerotic tissue. Figs. 247-249, Heidenhain's iron-haematoxylin.

Fig. 248, $\times 150$. Disappearance, atrophy, and rounding of cells in the hypoglossal nucleus, with marked glia cell reaction.

Fig. 249, $\times 200$. One rounded atrophic ganglion cell present in the midst of a dense glia network.

Fig. 250, $\times 200$. Ganglion cells in one of the posterior root ganglia related to a completely demyelinated area. Polychrome methylene blue.

PLATE XLIII.

Figs. 251-262. Changes related to the axis cylinders.

Fig. 251, $\times 10$. Intersection in the median raphe of the pons of persistent axis cylinders. Note the darker staining of the still myelinated tissue on both sides (*cf.* figs. 16, 17, 44, and 119).

Fig. 252, $\times 300$. Persistent swollen axis cylinders in a medullary ray.

Fig. 253, $\times 200$, and fig. 254, $\times 50$. Persistent axis cylinders continued as shadowy lines into the dense sclerotic tissue; longitudinal sections of cord. Figs. 251-254, Bielschowsky-Williamson silver method.

Figs. 255 and 256, $\times 300$. Granular disintegration of the axis cylinders in a sclerosing area. Haematoxylin and eosin.

PLATE XLIV.

Figs. 257 and 258, $\times 200$. Persistent swollen axis cylinders in cross section; posterior columns of the cord. Bielschowsky-Williamson silver method.

Fig. 259. Complete retention of axis cylinders; longitudinal section of the cord. Figs. 259-262, Cajal's silver method. $\times 200$.

Fig. 260. Small area in cerebral white matter; axis cylinder network.

Figs. 261 and 262. Axis cylinder contact in optic chiasma and nerves which were completely gelatinous.

PLATE XLV.

Figs. 263-274. Sequence of changes in the blood-vessels (pp. 119-122); *cf.* figs. 13-15.

Fig. 263. Area in longitudinal section of the spinal cord showing capillary and transition vessels with rows of fat granule cells (*c*) in their adventitia. Van Gieson's stain. $\times 60$.

Figs. 264-267. Blood-vessels in the cerebral white matter. Heidenhain's iron-haematoxylin. $\times 200$. Fig. 264, vessel cut transversely (*b*) with fat granule cells (*c*) in its adventitial lymph spaces, and glia cell reaction (*a*) in the surrounding tissue; fig. 265, $\times 200$, similar vessel cut longitudinally; fig. 266, $\times 400$, H.P. of previous figure to show the relation of the glia "fuss" to the outer layers of the adventitia; fig. 267, $\times 75$, vessel surrounded by concentric layers of glia fibrils.

Fig. 268. Vessel in the posterior median fissure; adventitial lymph spaces filled by fat granule cells which have been drained from the surrounding sclerotic tissue. Van Gieson's stain, $\times 250$.

PLATE XLVI.

Fig. 269. (*cf.* fig. 14). Transition vessel to show the cell content of the adventitia during the stage of advancing sclerosis. Van Gieson's stain, $\times 370$.

Fig. 270. Condensed "hyaline" vessel (*a*) with the outer layers (*b*) of its adventitia still separated. Note lessening cell content of the vessel wall. Iron-haematoxylin. $\times 360$.

Fig. 271. Similar vessels with the peri-vascular spaces (artefacts) filled with a coagulum (*a*). Van Gieson's stain. $\times 70$.

Figs. 272, $\times 60$. Lateral columns of the spinal cord; radiating longitudinal thickened vessels (*a*). Figs. 272-274, Kulschitzky-Pal with picro-fuchsin.

Fig. 273, $\times 50$. Posterior columns of the spinal cord with similar vessels cut transversely (*a*) and longitudinally (*b*).

Fig. 274, $\times 250$. Optic nerve; connective-tissue septa and thickened vessels in longitudinal section.

PLATE XLVII.

Figs. 275-280. Special features of the changes in the blood-vessels.

Fig. 275. Cerebral white matter; small dilated capillary surrounded by a zone of shadow sclerosis. Kulschitzky-Pal with picro-fuchsin. $\times 25$.

Fig. 276. Cerebral cortex; two dilated and thickened small cortical vessels passing into a demyelinated area. Van Gieson's stain. $\times 50$.

Fig. 277. Dilated vessels at posterior horn of lateral ventricle. Van Gieson's stain. $\times 50$.

Fig. 278. Groups of vessels in a sclerotic area with very dilated peri-vascular tissue. Kulschitzky-Pal with picro-fuchsin. $\times 10$.

Fig. 279. Similar vessels in an area of more advanced sclerosis. Van Gieson's stain. $\times 30$.

Fig. 280. Similar vessels both within the area and in the adjoining tissue. Van Gieson's stain. $\times 45$.

PLATE XLVIII.

Figs. 281-284. Illustrate various features in the changes in the nerve roots. Kulschitzky-Pal with picro-fuchsin.

Fig. 281, $\times 75$. Rarefaction of the anterior nerve roots.

Fig. 282, $\times 75$. Normal nerve roots in the cauda equina. H.P. of next figure.

Fig. 283, $\times 6$. Normal nerve roots surrounding a completely demyelinated fifth sacral segment of the cord.

Fig. 284, $\times 6$. Normal sciatic nerve in same case as previous figure.

Fig. 285. Ependymal proliferation in wall of lateral ventricle. Heidenhain's iron-haematoxylin. $\times 300$.

Fig. 286. Normal glandular portion of the pituitary body. Haematoxylin and eosin. $\times 300$.

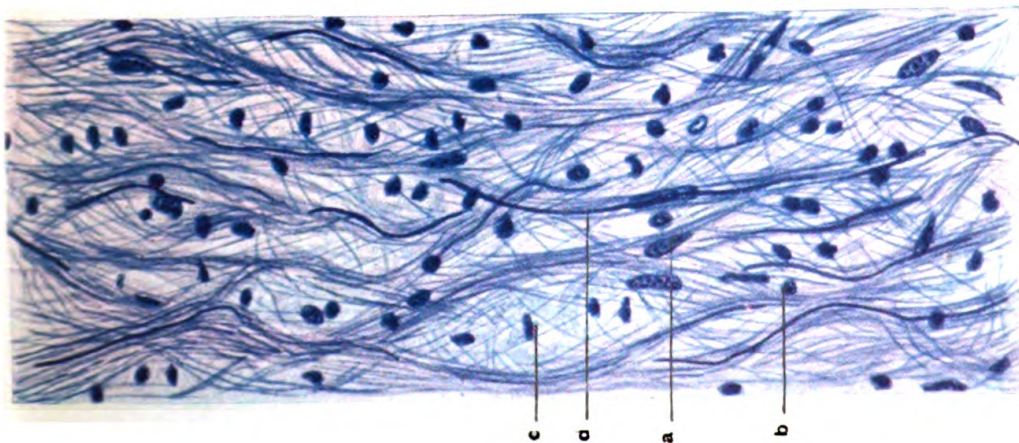


FIG. 1.

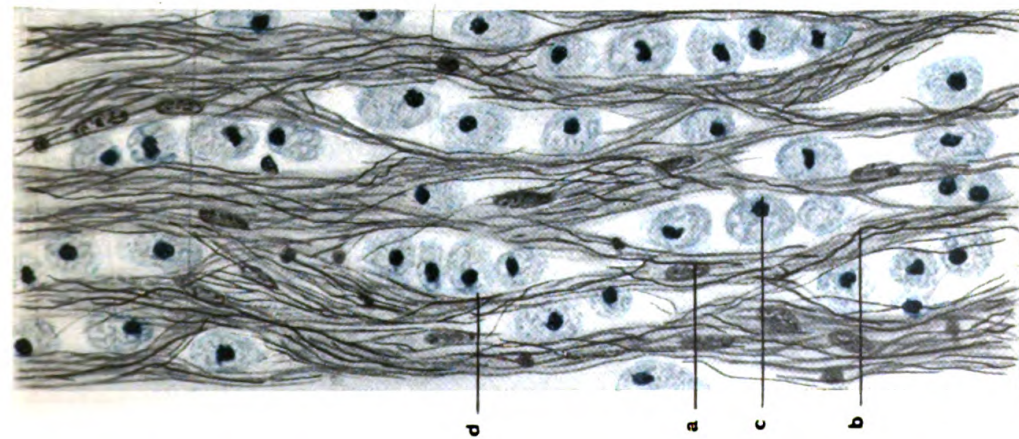


FIG. 2.

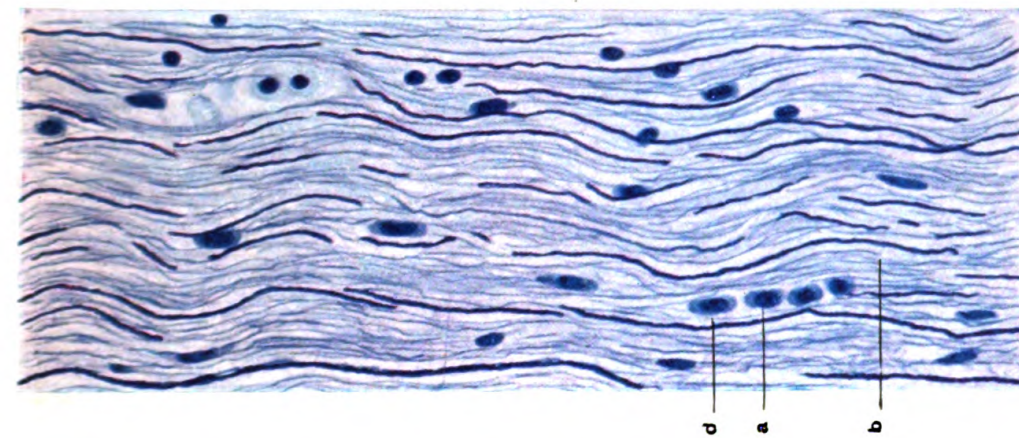


FIG. 3.

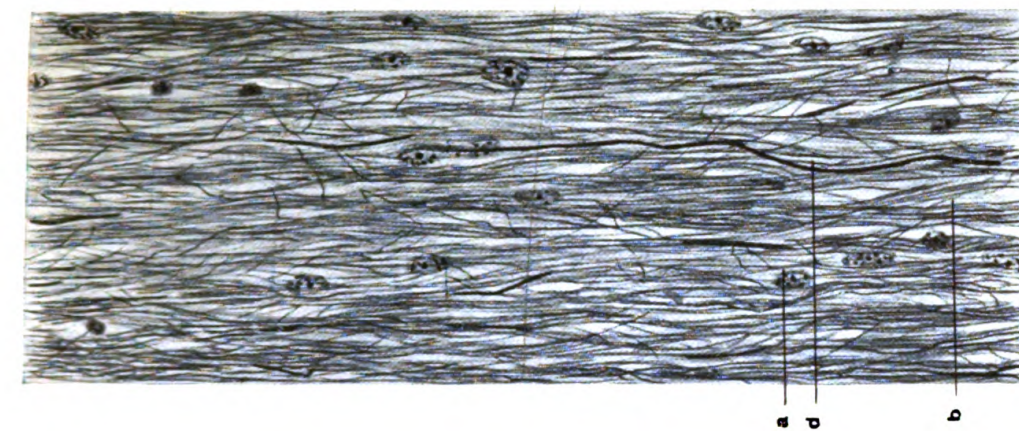


FIG. 4.

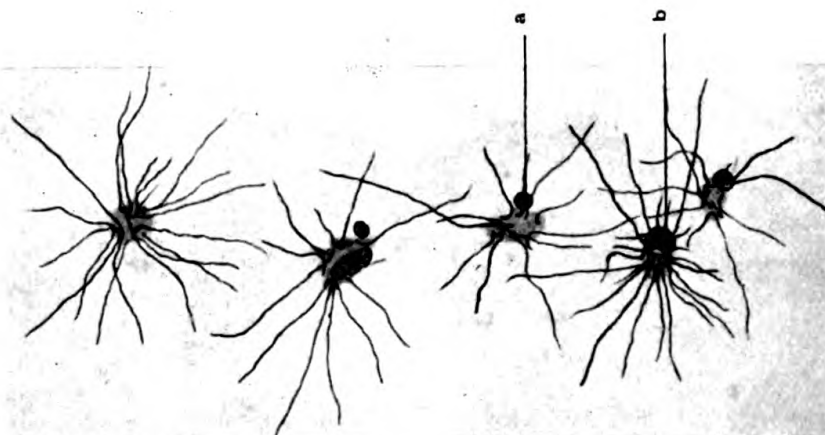


FIG. 7.

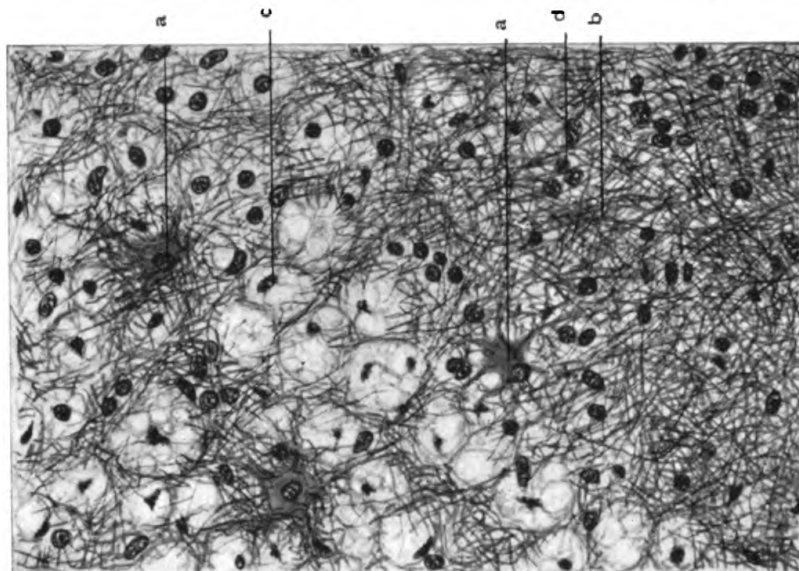


FIG. 6.

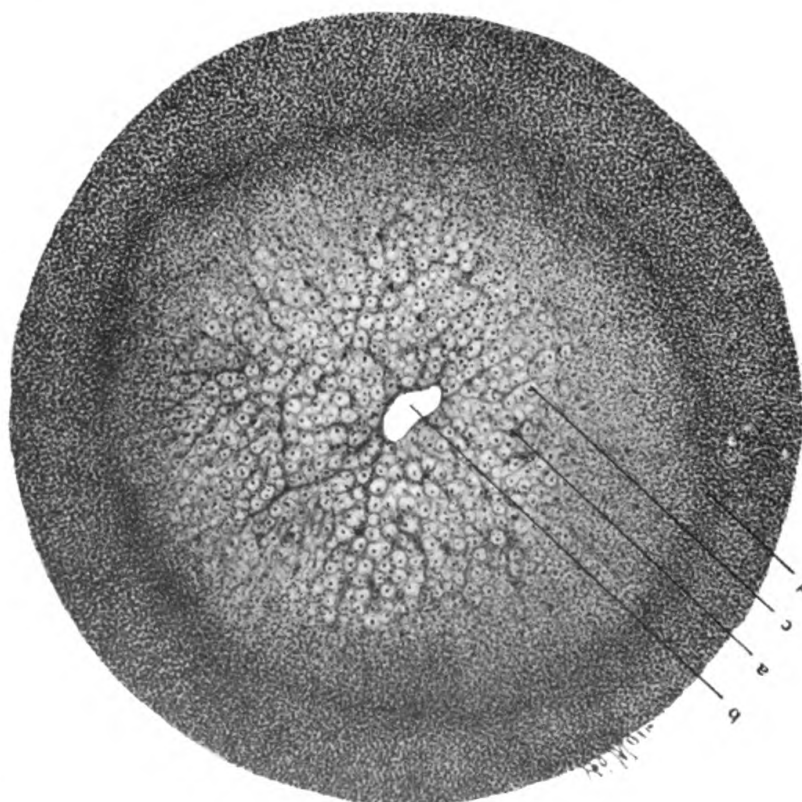


FIG. 5.

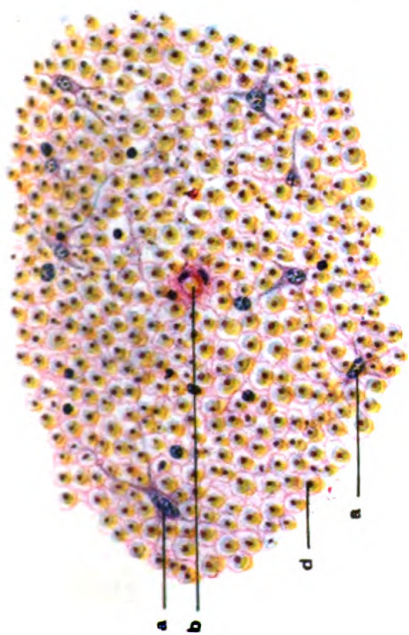


FIG. 8.

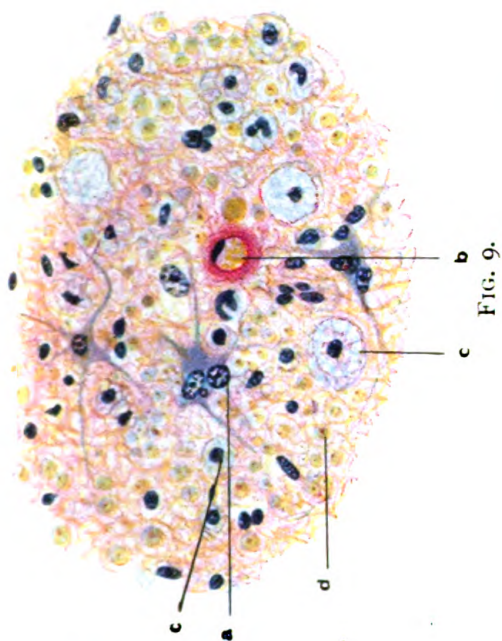


FIG. 9.

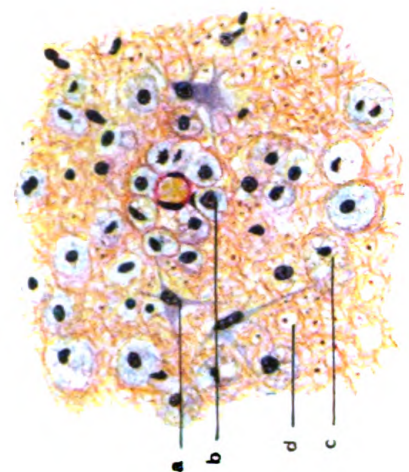


FIG. 10.



FIG. 11

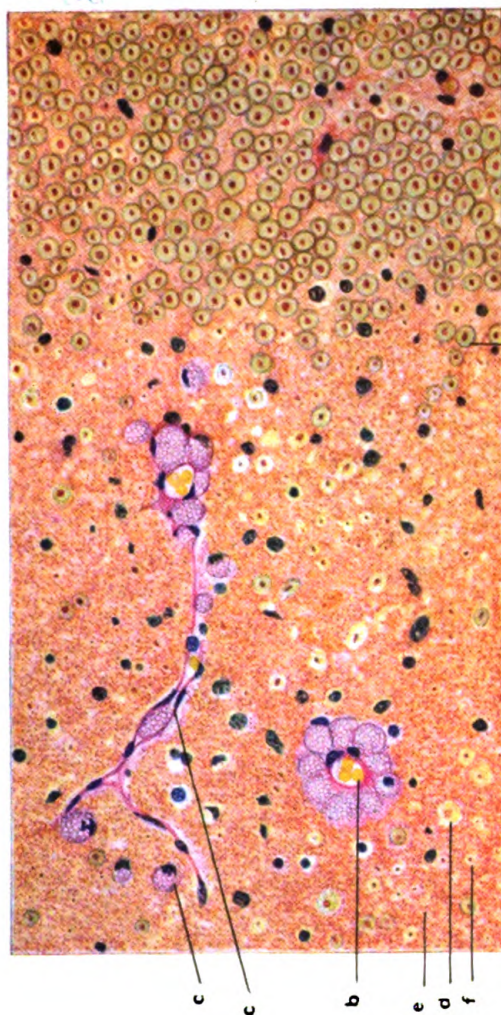


FIG 12. g

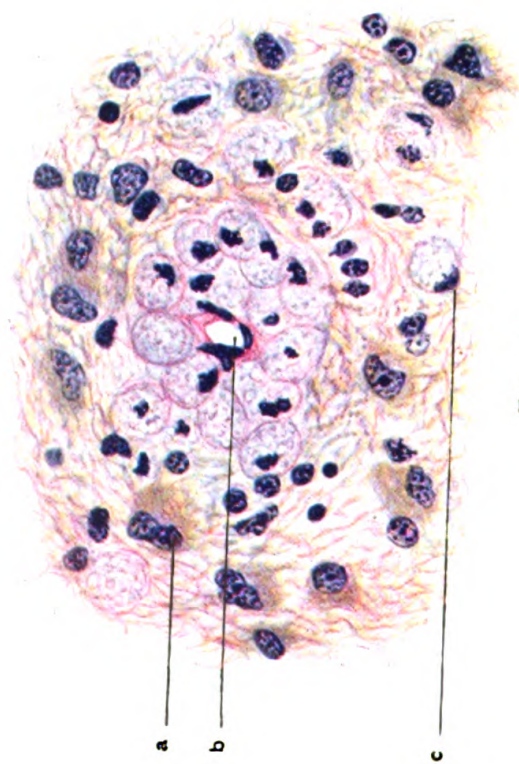


FIG. 13.

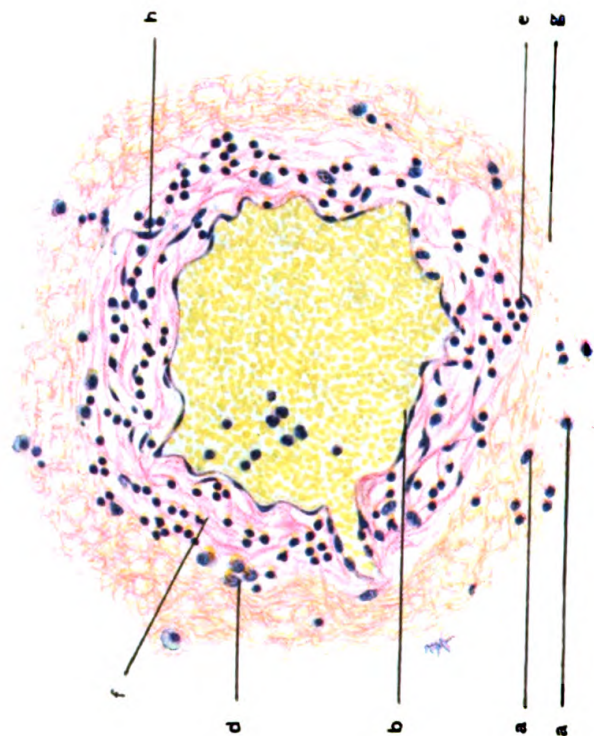


FIG. 14.



FIG. 15.



FIG. 16.



FIG. 17.

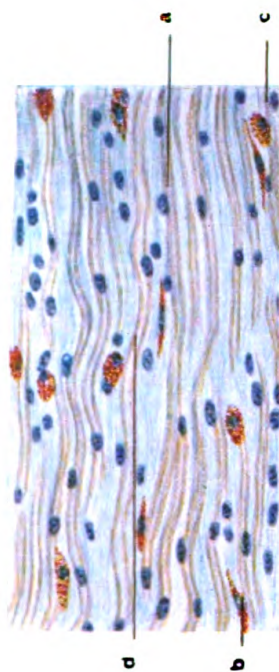


FIG. 18.

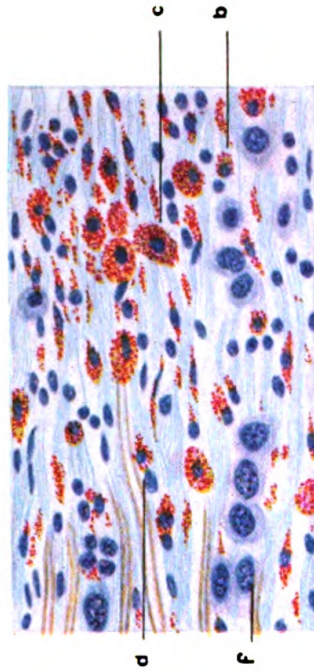


FIG. 19.

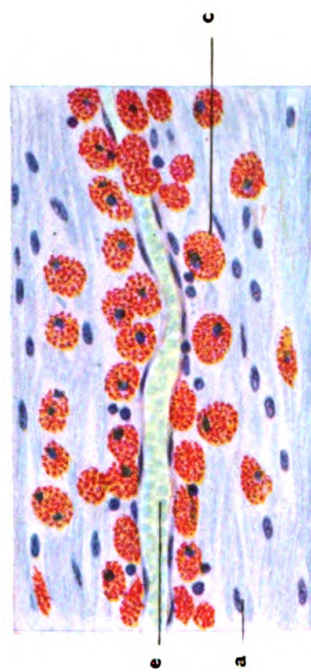


FIG. 20.

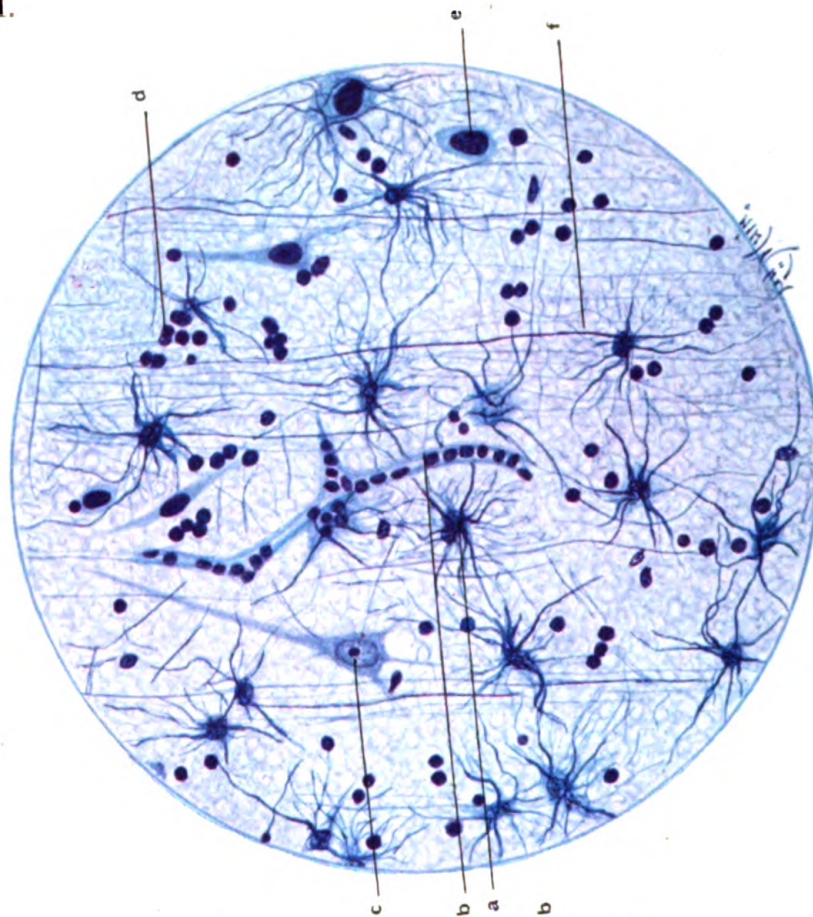


FIG. 22.

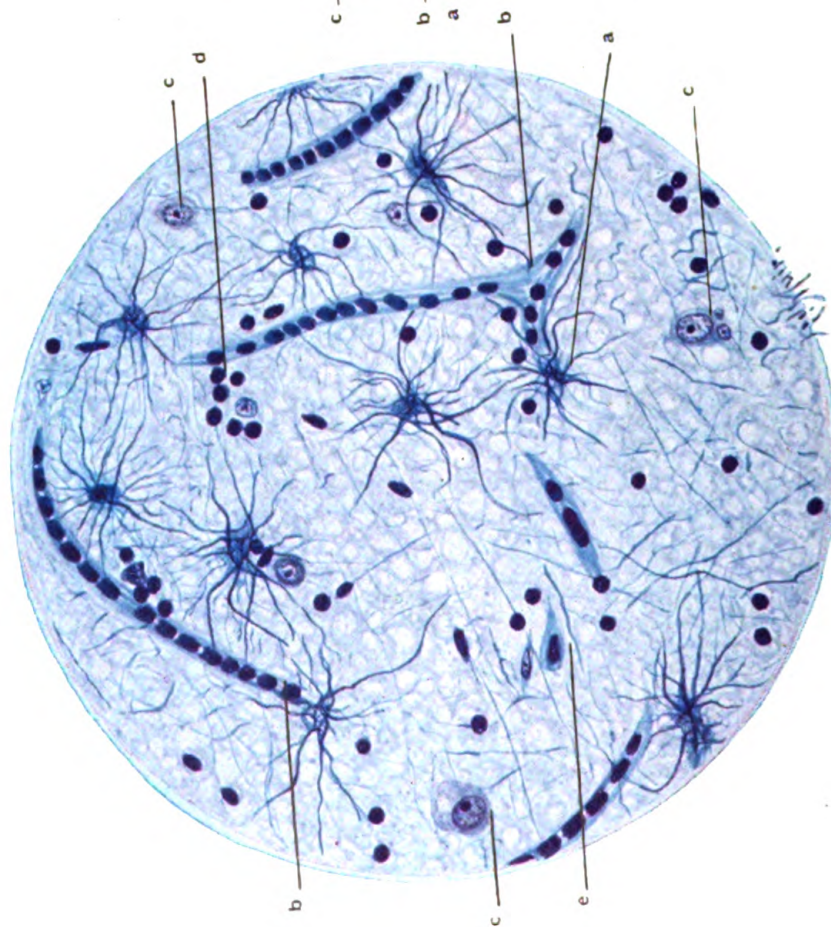


FIG. 21.

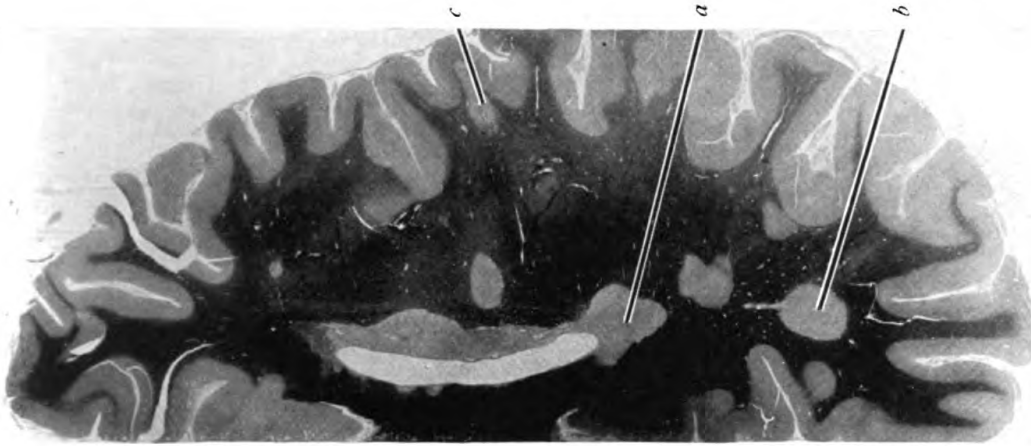


FIG. 26.

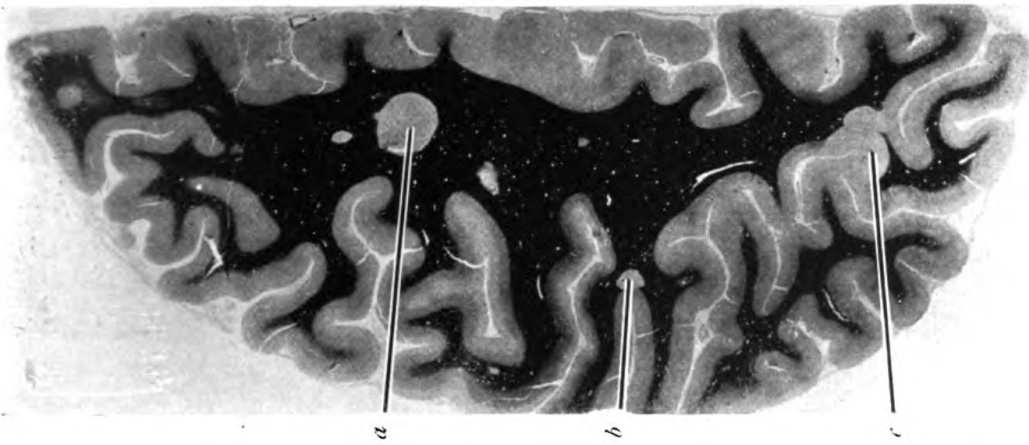


FIG. 25.

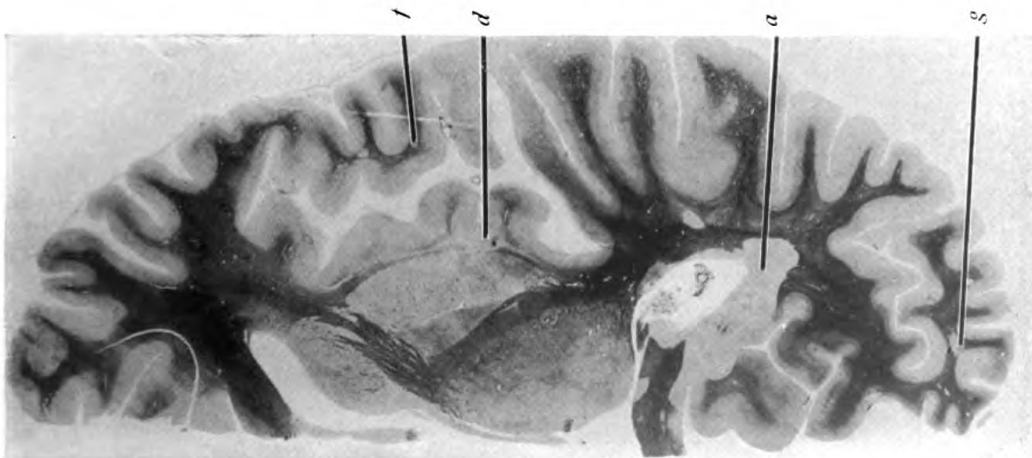


FIG. 24.

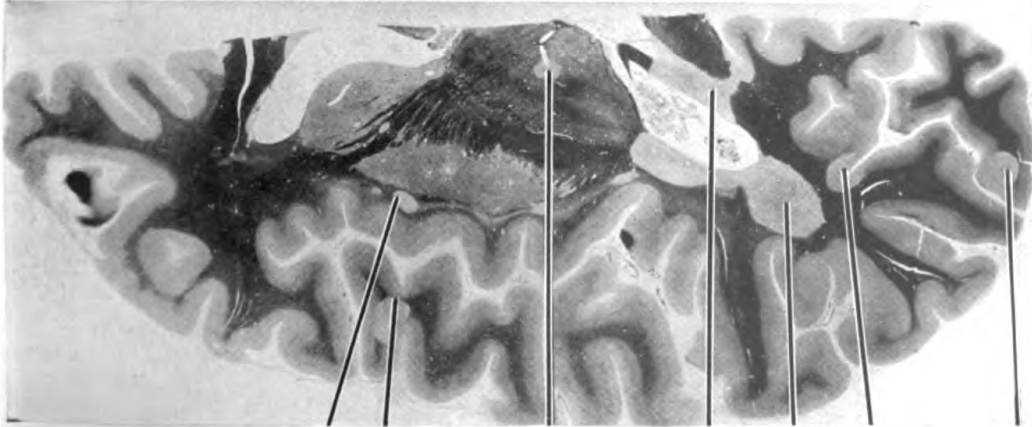


FIG. 23.

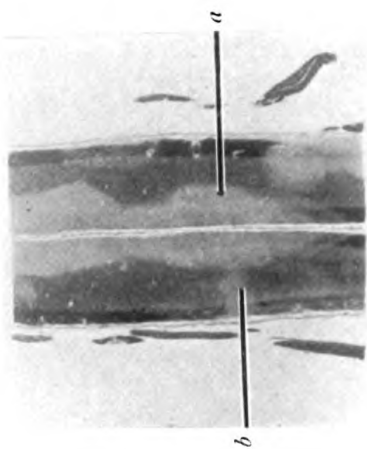


FIG. 30.

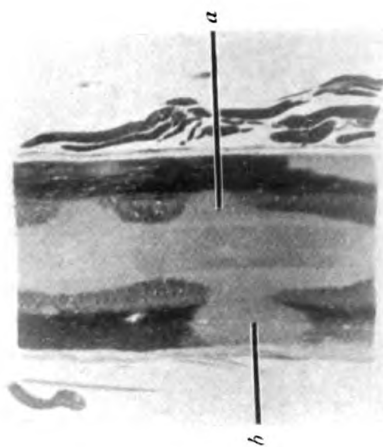


FIG. 31.

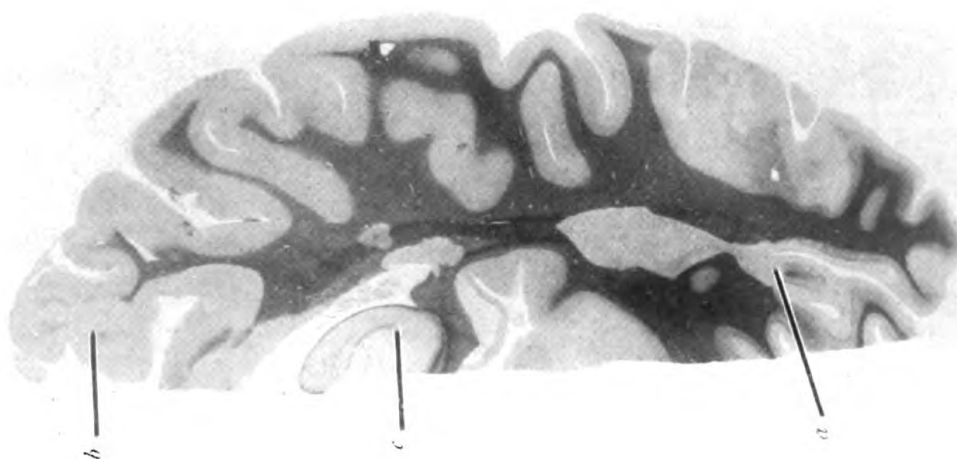


FIG. 29.

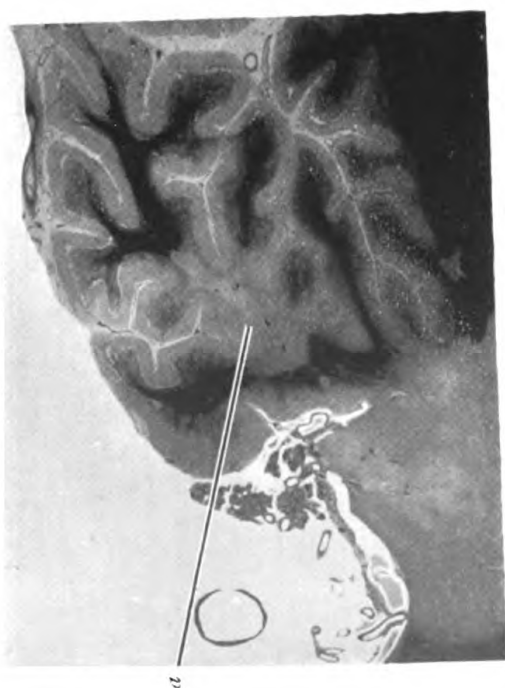


FIG. 27.



FIG. 28.

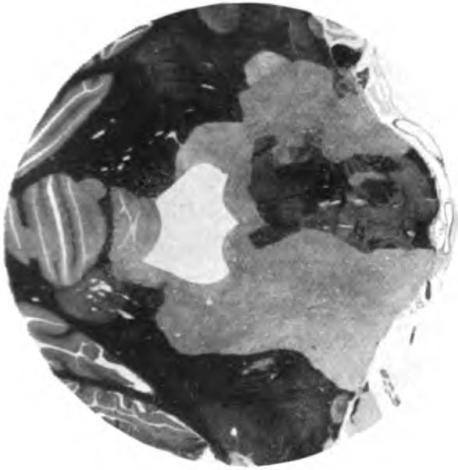


FIG. 40.



FIG. 41.

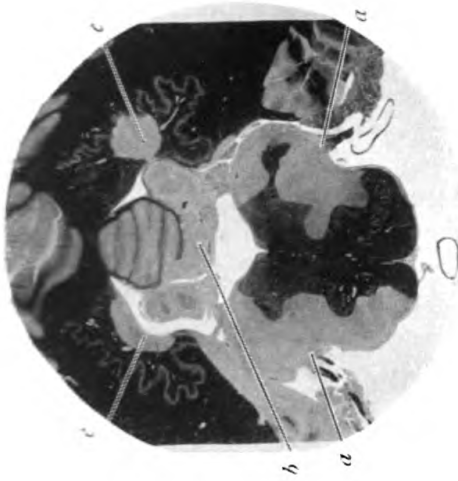


FIG. 38.



FIG. 39.

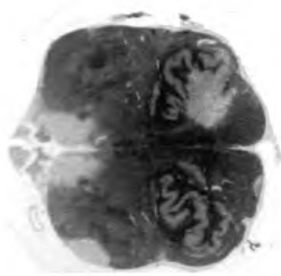


FIG. 35.

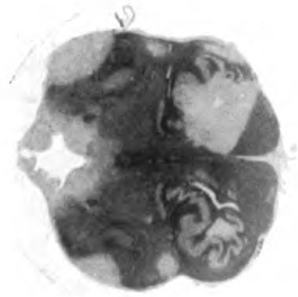


FIG. 36.

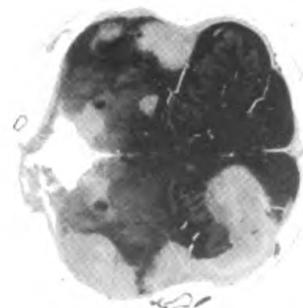


FIG. 37.

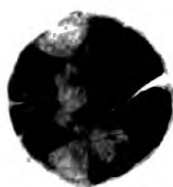


FIG. 32.



FIG. 33.



FIG. 34.



FIG. 46.

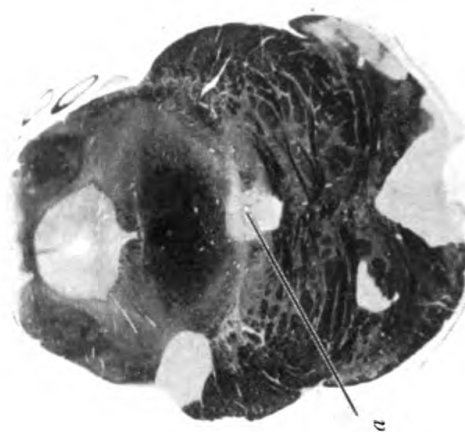


FIG. 47.



FIG. 44.

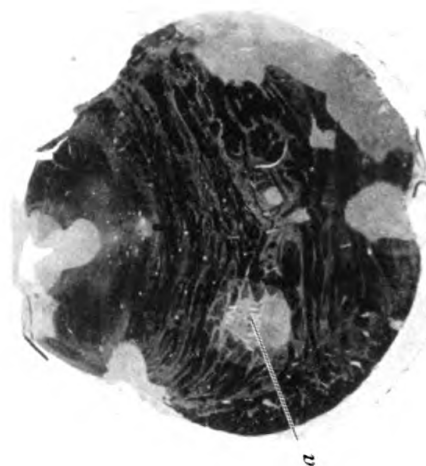


FIG. 45.



FIG. 42.



FIG. 43.



FIG. 48—Upper C₂

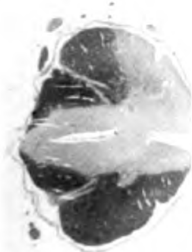


FIG. 49—Lower C₂

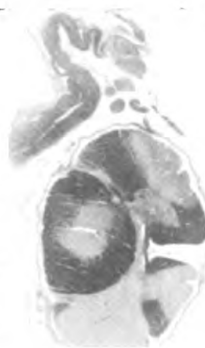


FIG. 50—Upper C₃



FIG. 51—Lower C₃



FIG. 52—C₃

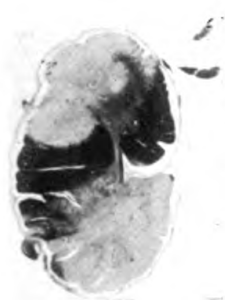


FIG. 53—C₆



FIG. 54—Upper C₈

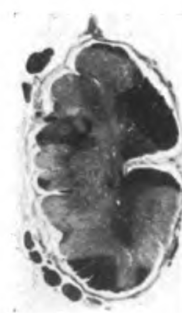


FIG. 55—D₁



FIG. 56—D₄



FIG. 57—D₆

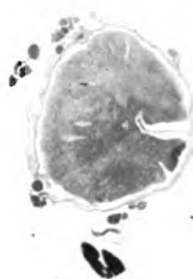


FIG. 58—D₁₀



FIG. 59—L₃



FIG. 60—L₄



FIG. 61—S₁

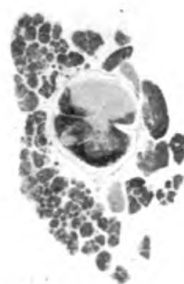


FIG. 62—S₂



FIG. 63—S₅

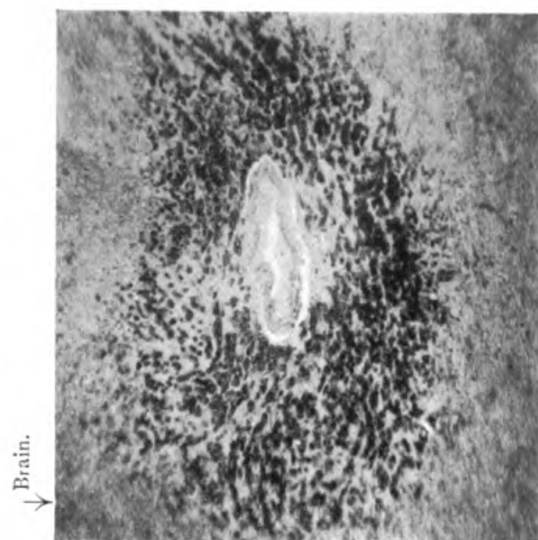


FIG. 68.

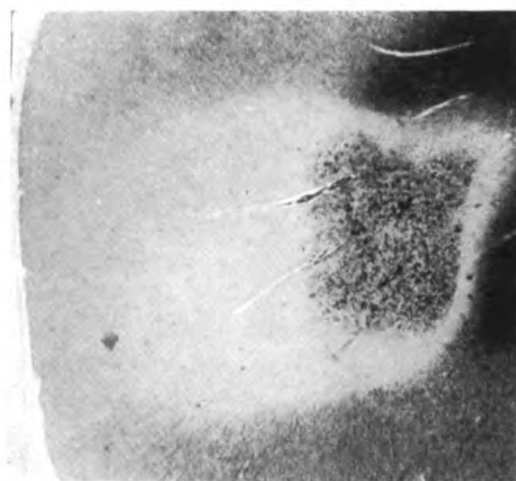


FIG. 69.



FIG. 66.

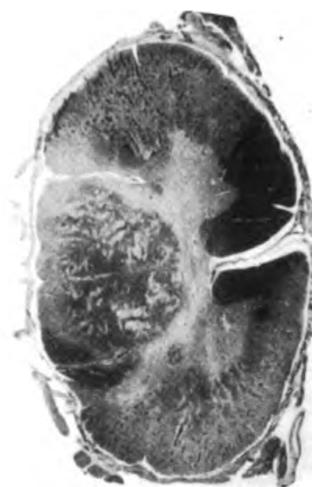


FIG. 67.

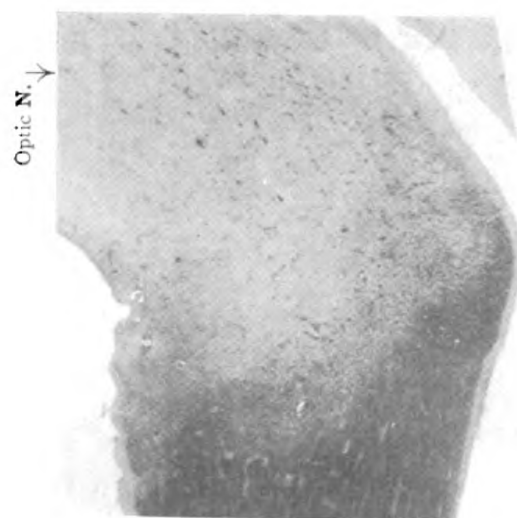


FIG. 64.



FIG. 65.



FIG. 72.

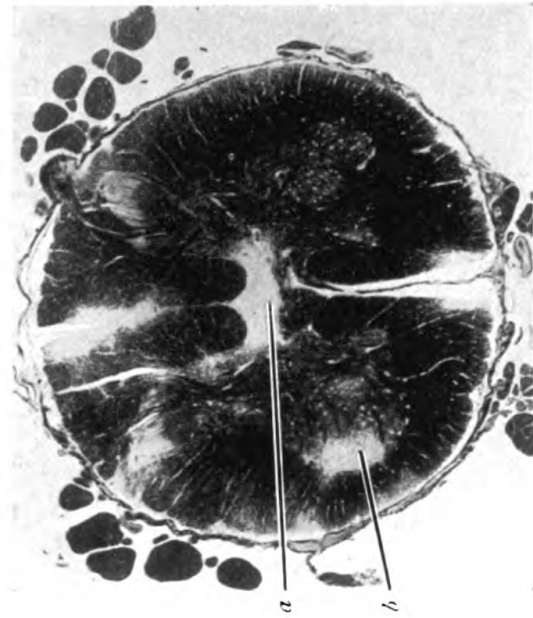


FIG. 73.



FIG. 70.

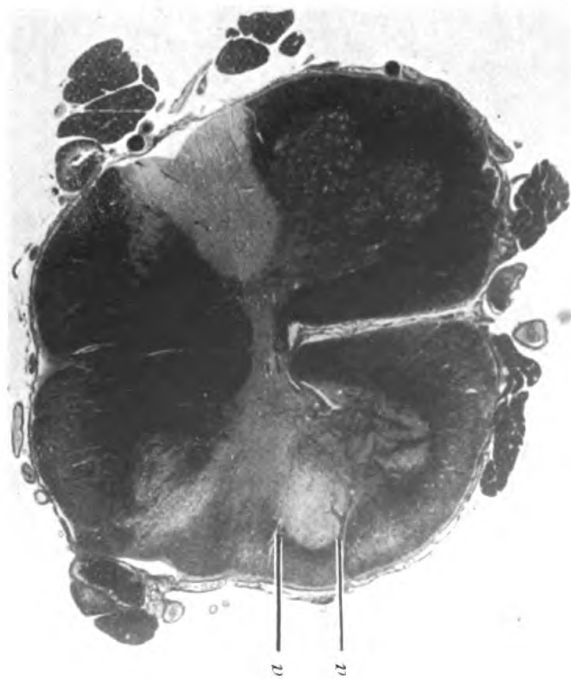


FIG. 71.

PLATE XIV.

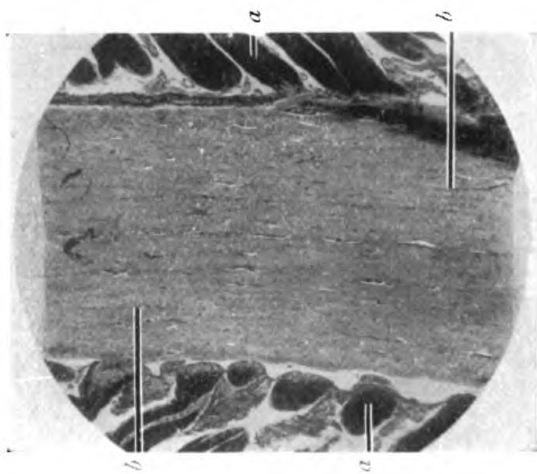


FIG. 80.



FIG. 81.



FIG. 82.



FIG. 77.

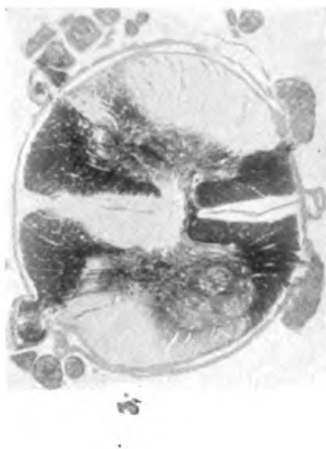


FIG. 78.

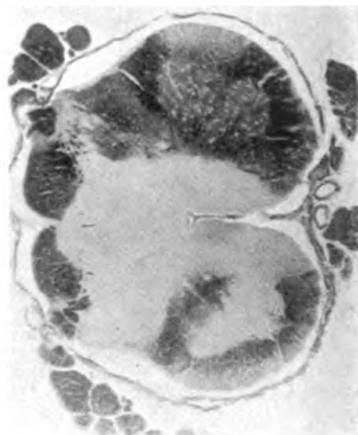


FIG. 79.



FIG. 74.



FIG. 75.



FIG. 76.

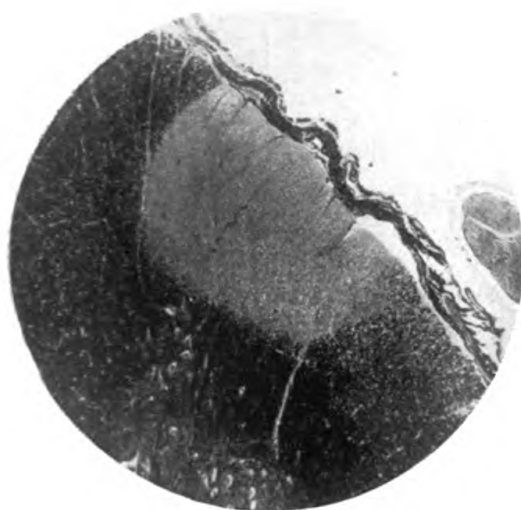


FIG. 87.

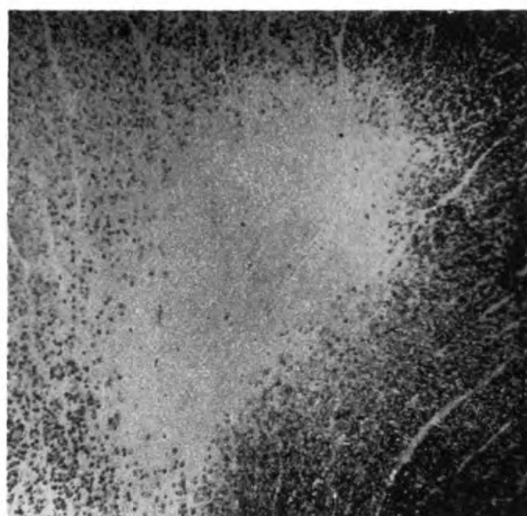


FIG. 88.

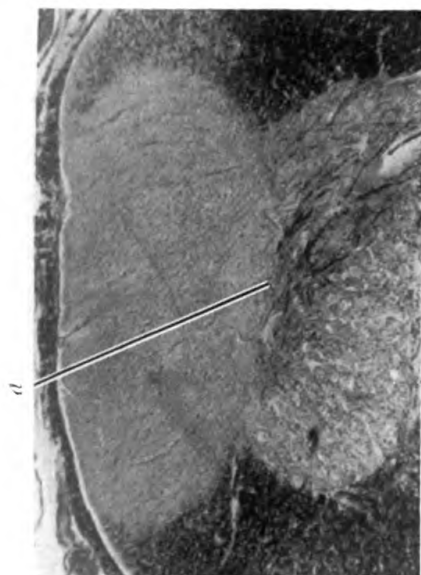


FIG. 85.

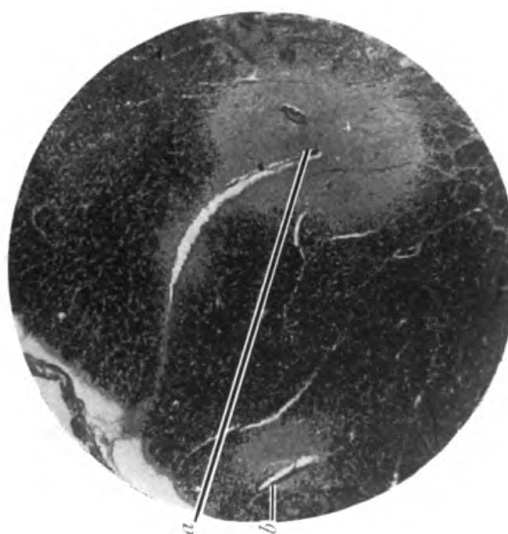


FIG. 86.



FIG. 83.

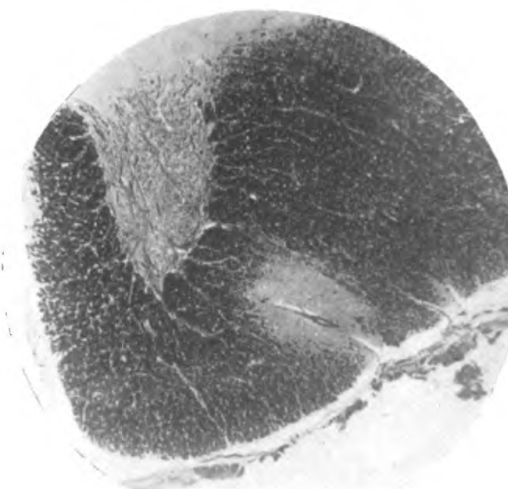


FIG. 84.

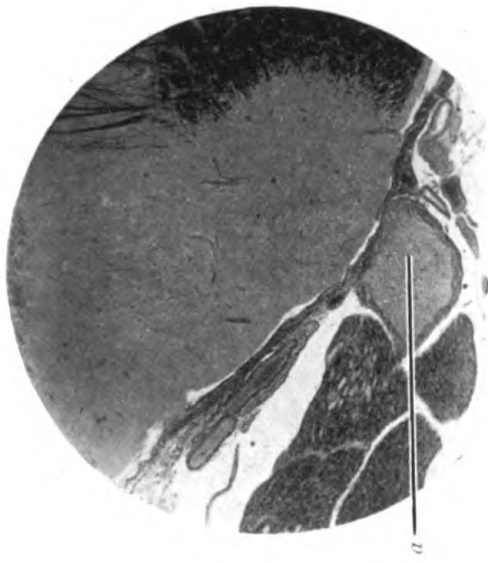


FIG. 93.



FIG. 94.

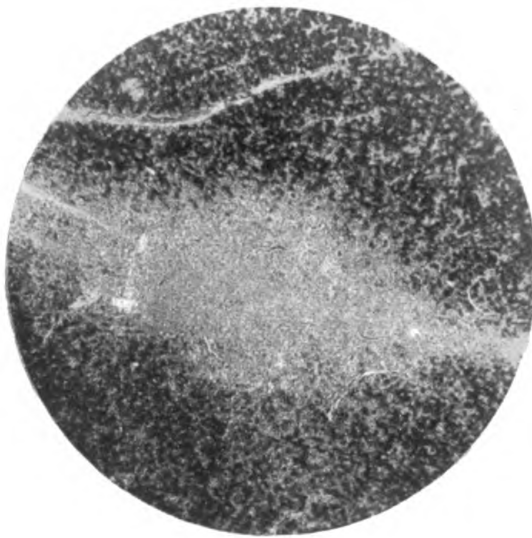


FIG. 91.

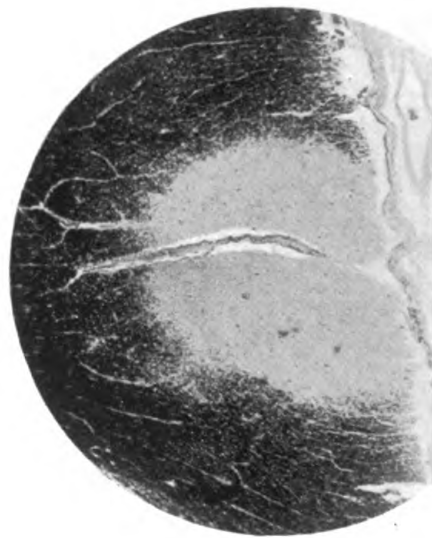


FIG. 92.

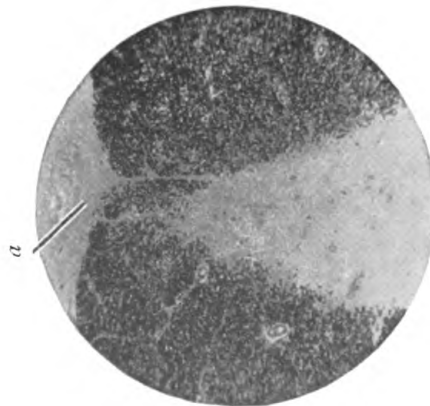


FIG. 89.

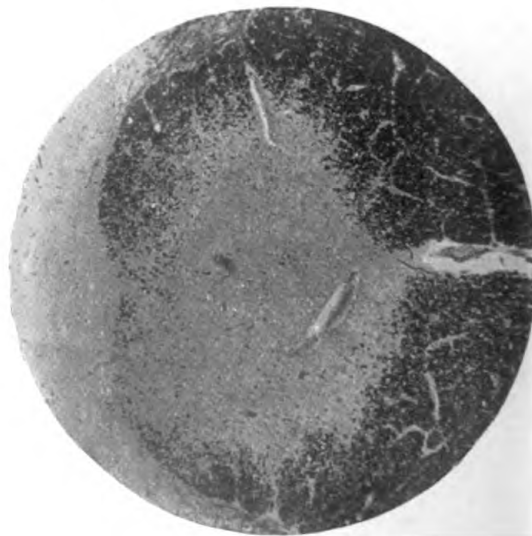


FIG. 90.

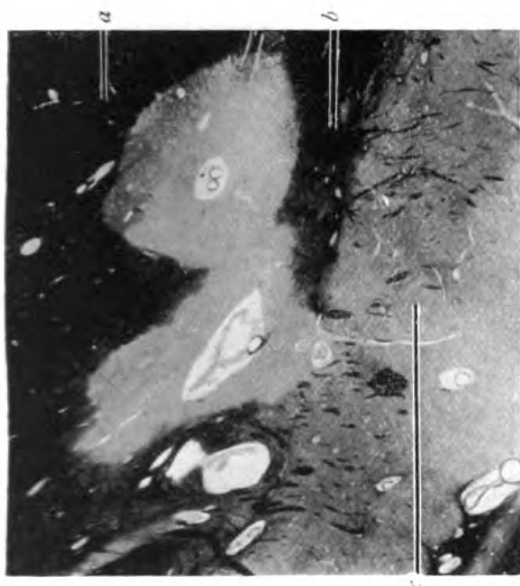


FIG. 97.

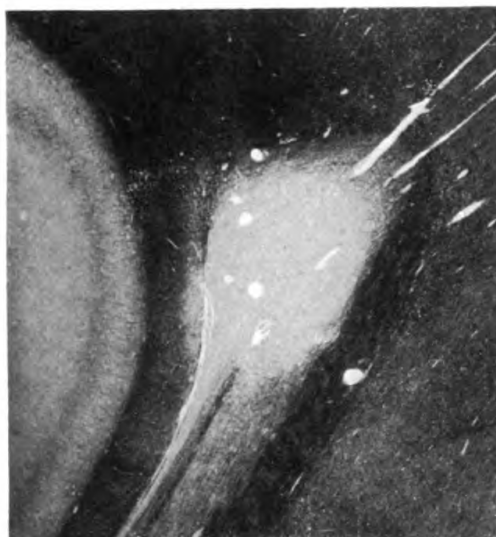


FIG. 100.

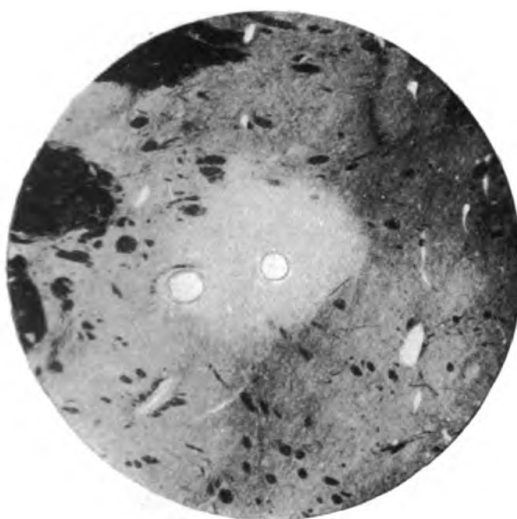


FIG. 96.



FIG. 99.

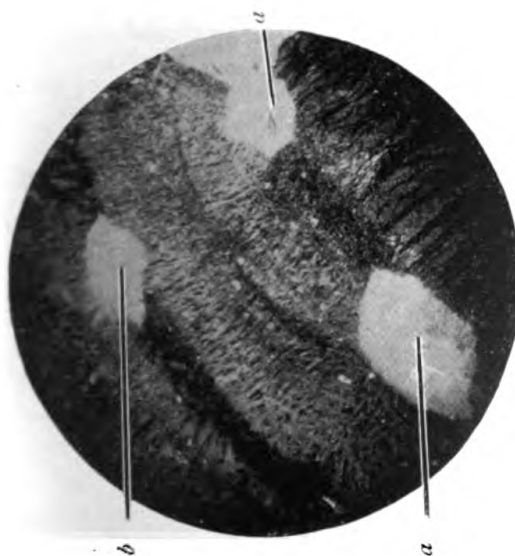


FIG. 95.

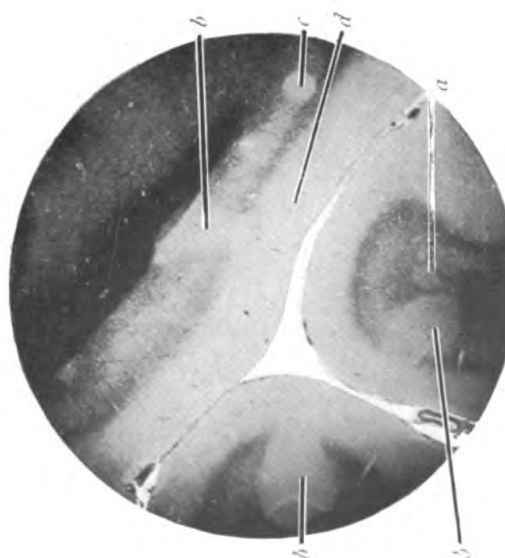


FIG. 98.

PLATE XVIII.

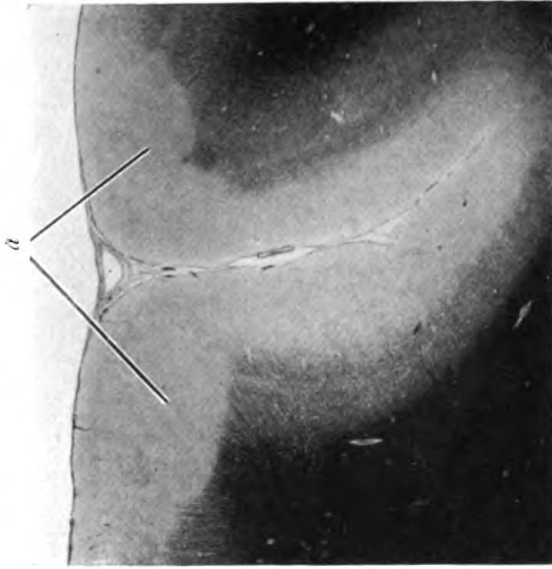


FIG. 103.

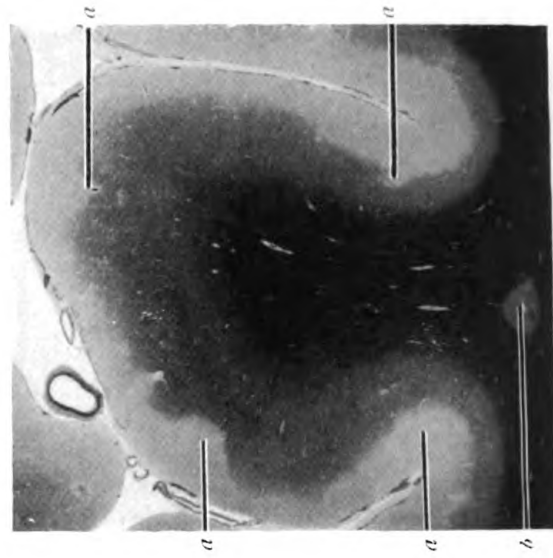


FIG. 100.

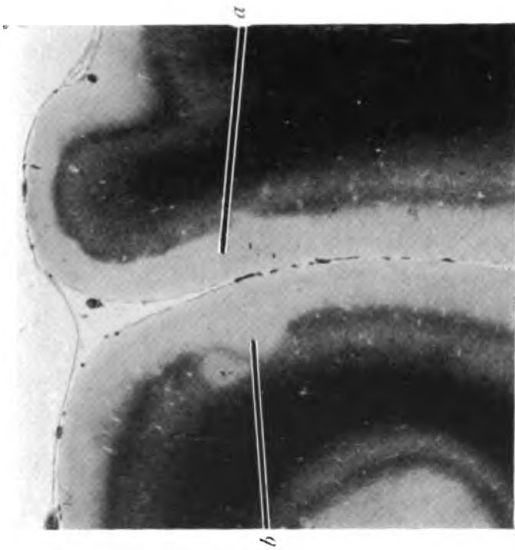


FIG. 102.

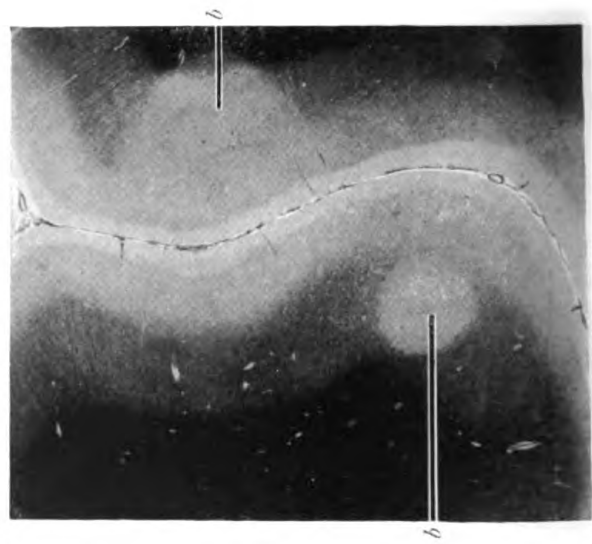


FIG. 105.

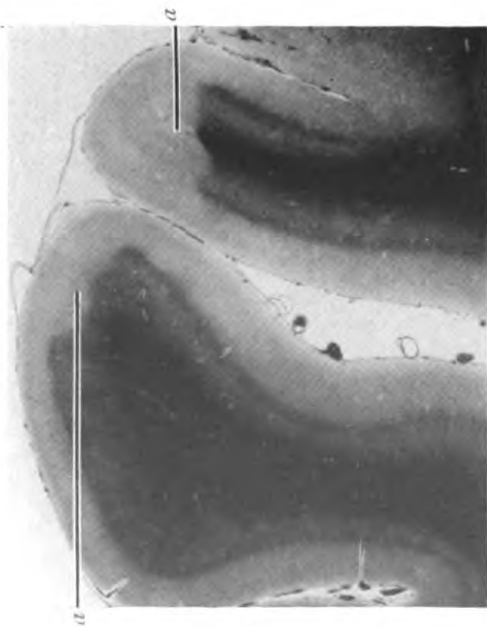


FIG. 101.

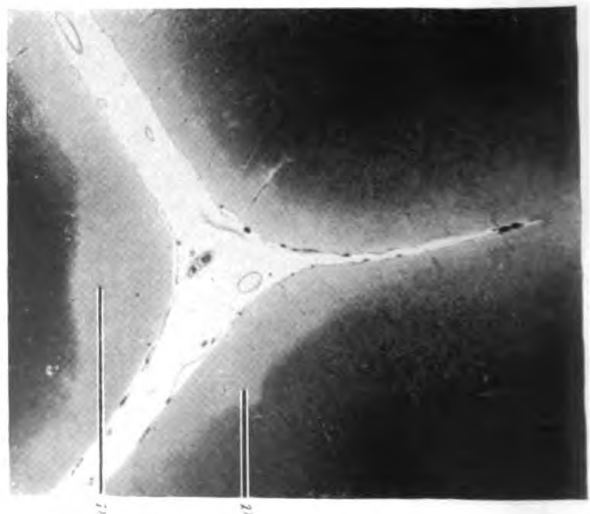


FIG. 104.

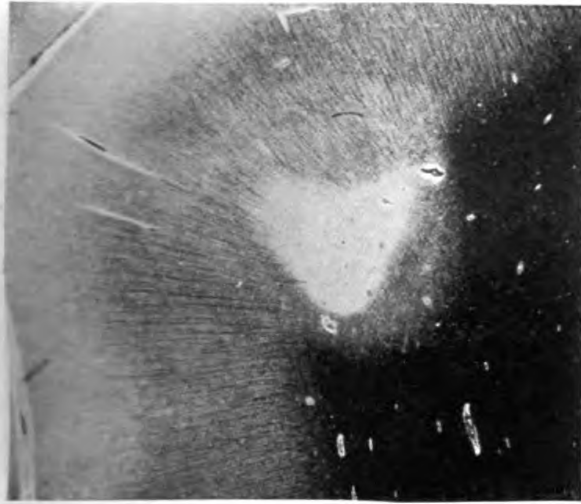


FIG. 109.

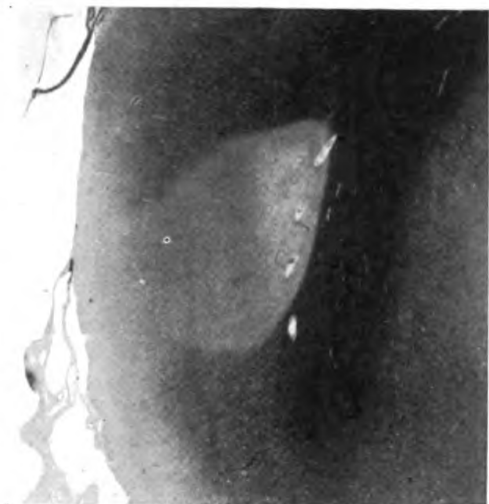


FIG. 112.

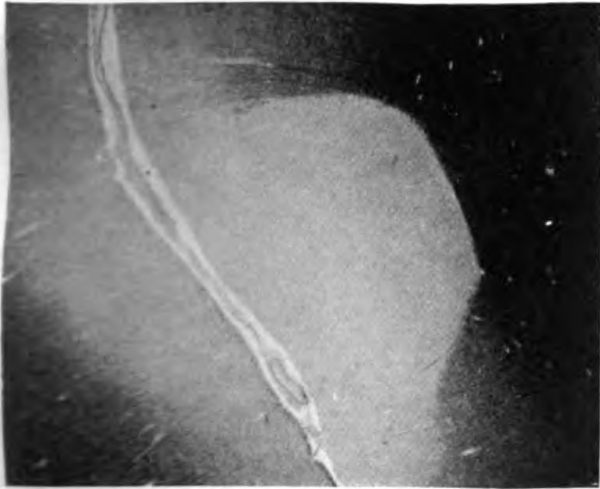


FIG. 108.

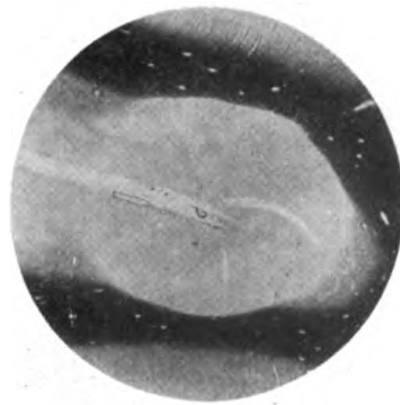


FIG. 111.



FIG. 107.

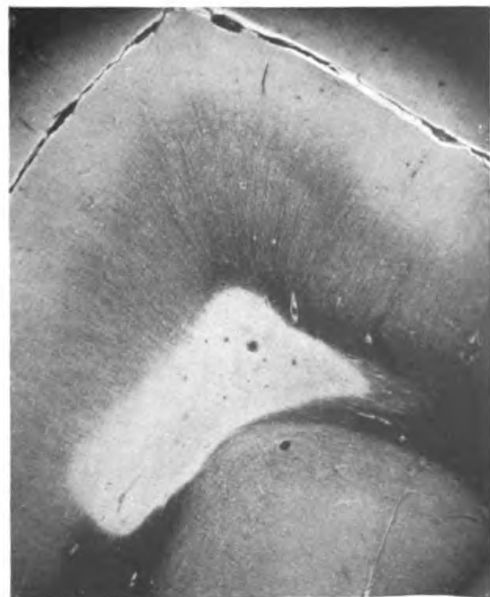


FIG. 110.

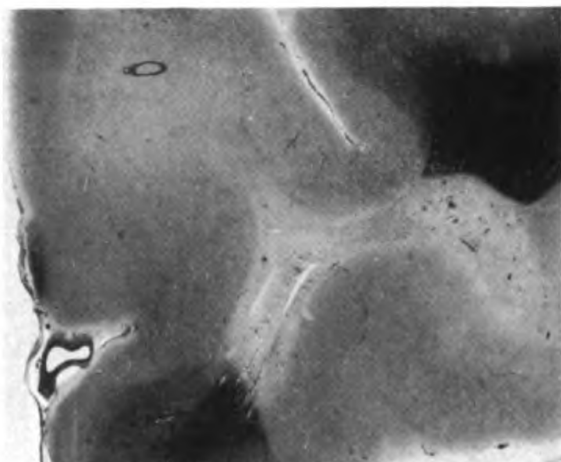


FIG. 115.

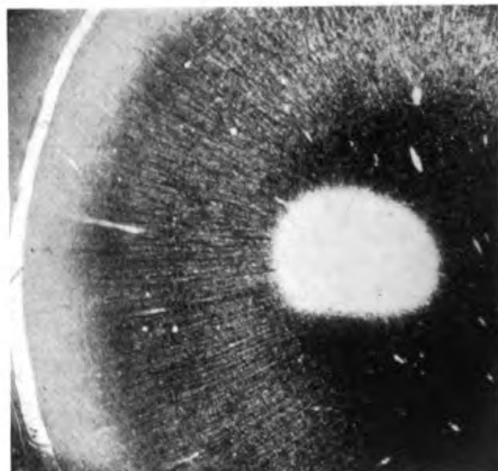


FIG. 118.



FIG. 114.



FIG. 117.

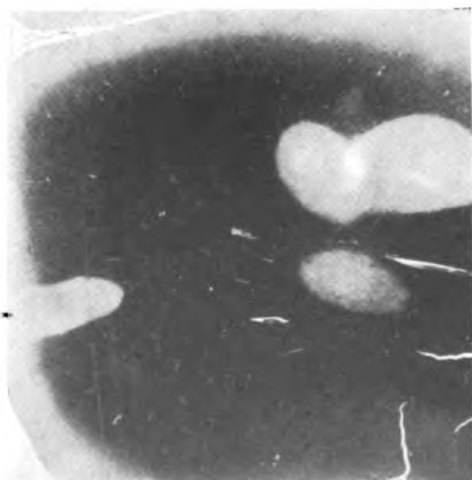


FIG. 113.

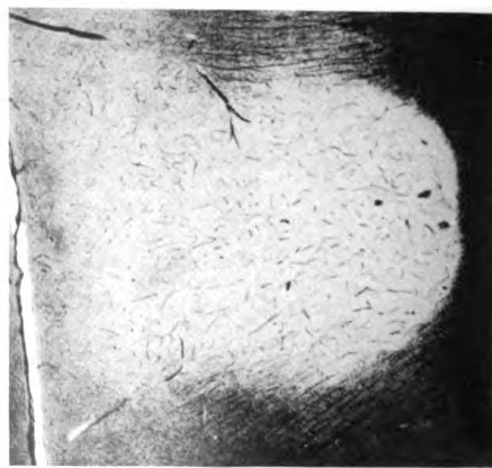


FIG. 116.

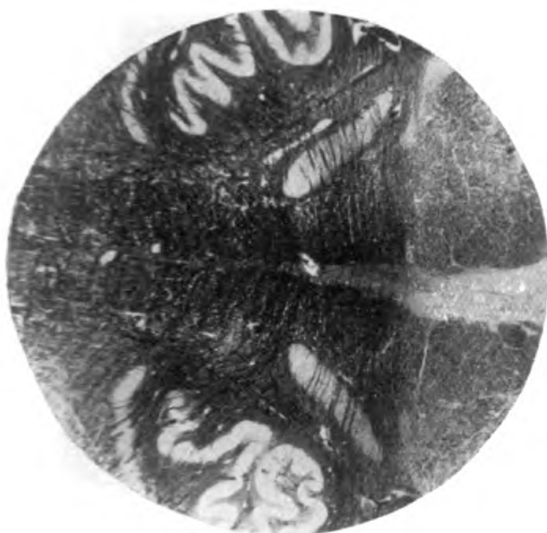


FIG. 121.

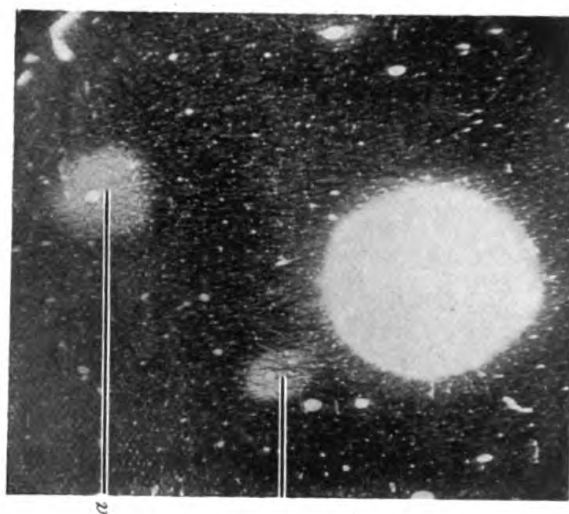


FIG. 124.

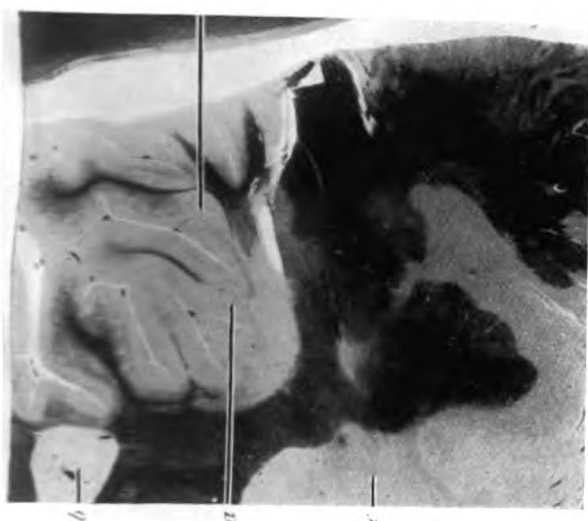


FIG. 120.

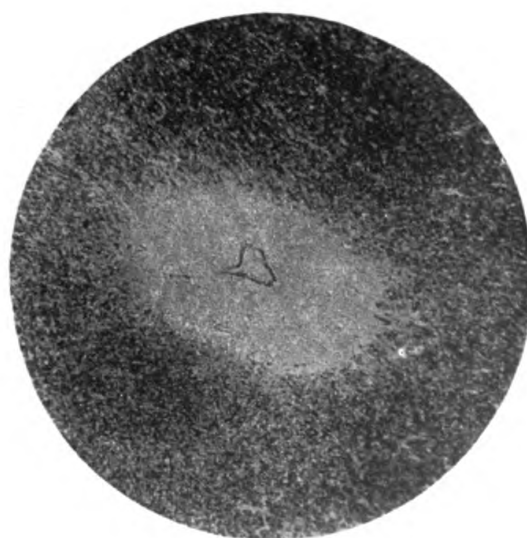


FIG. 123.

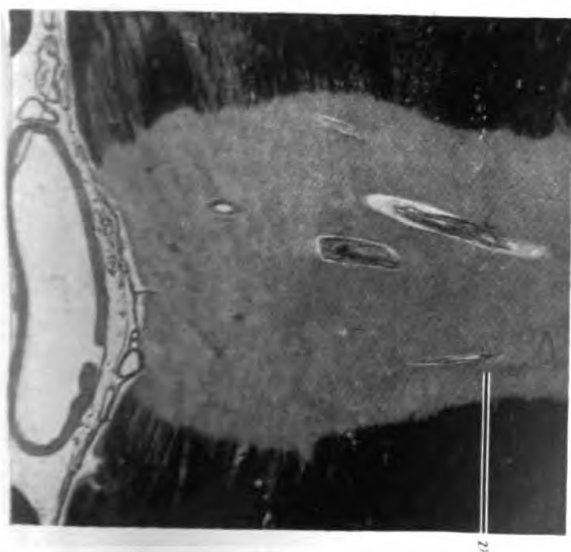


FIG. 119.



FIG. 122.

PLATE XXII.



FIG. 127.

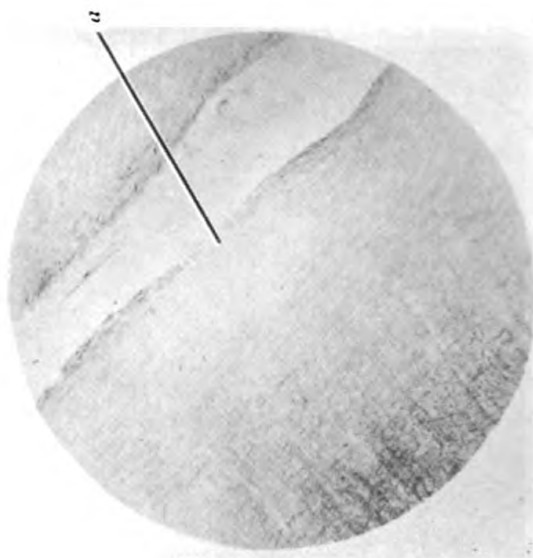


FIG. 130.

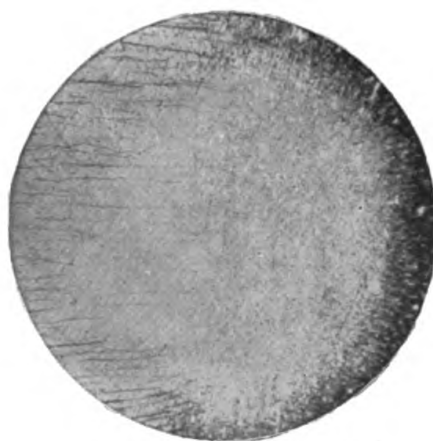


FIG. 126.

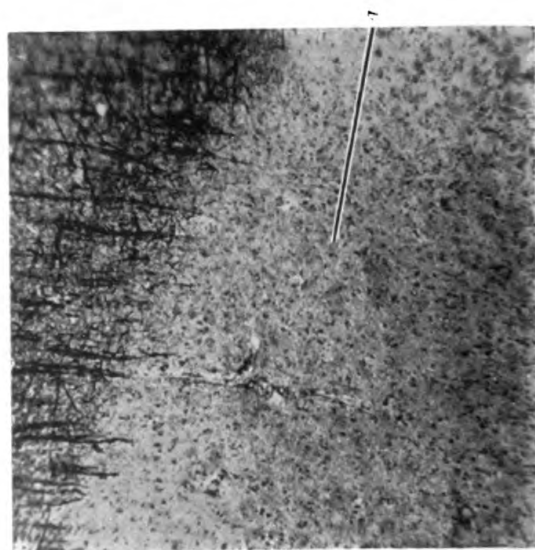


FIG. 129.



FIG. 125.

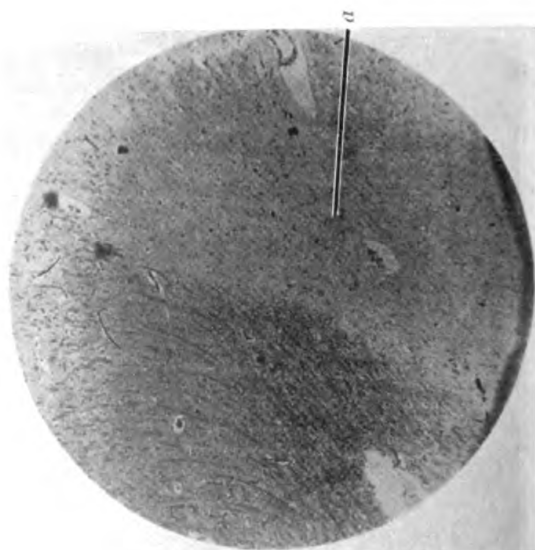


FIG. 128.



FIG. 133.

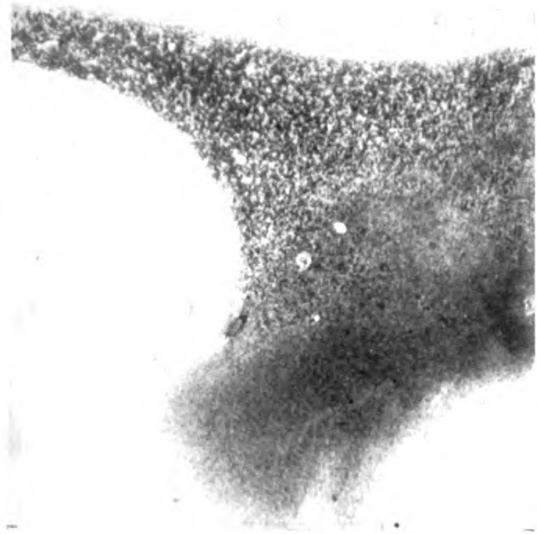


FIG. 136.

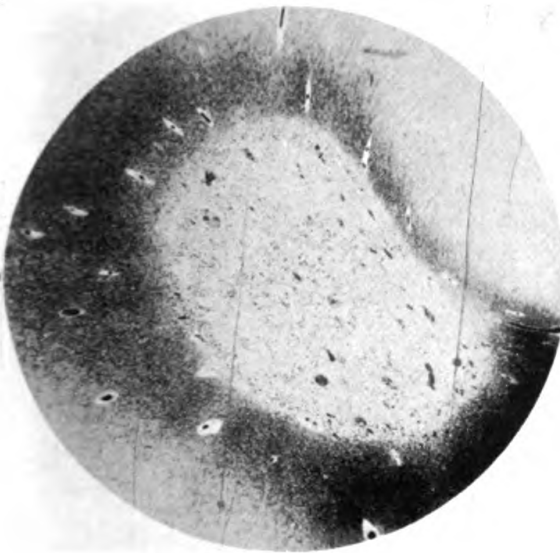


FIG. 132.



FIG. 135.

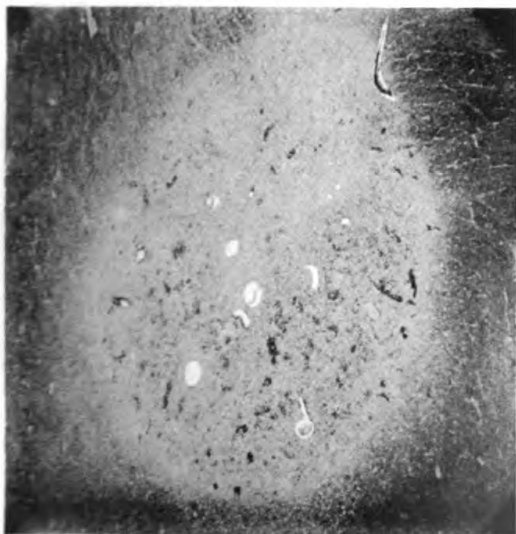


FIG. 131.

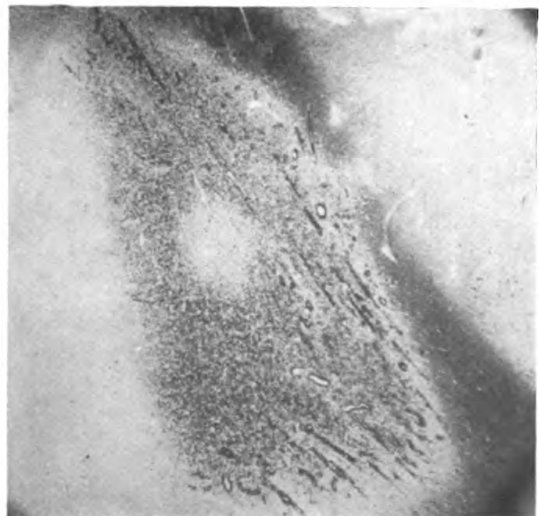


FIG. 134.

PLATE XXIV.

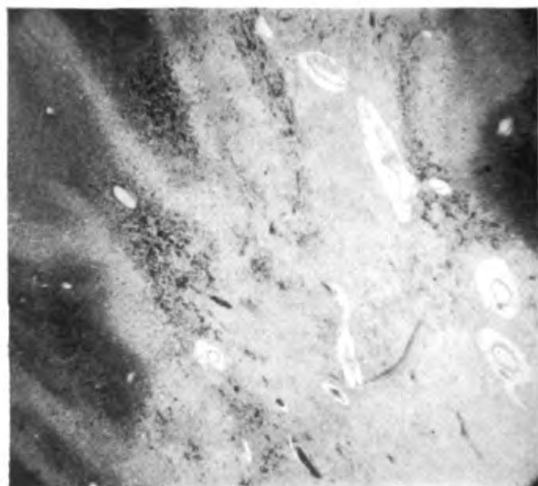


FIG. 139.

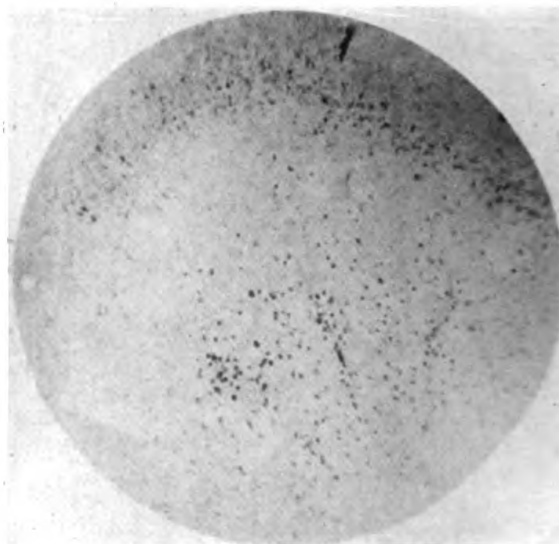


FIG. 142.



FIG. 138.

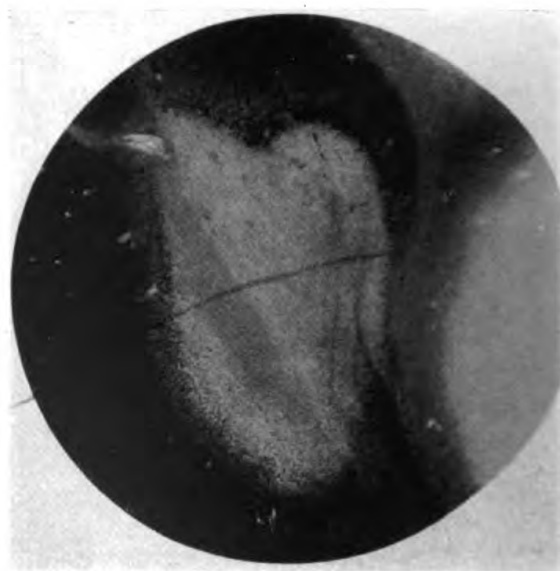


FIG. 141.

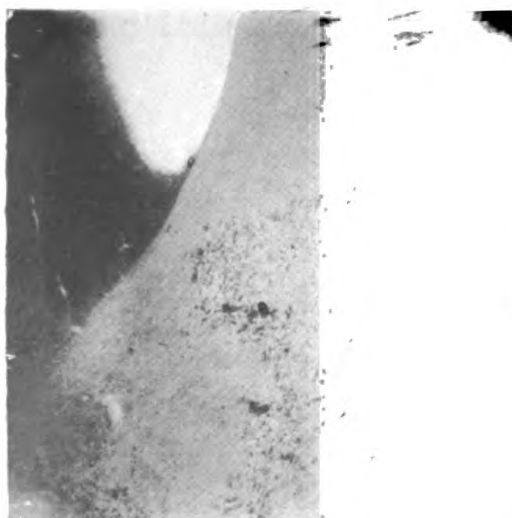


FIG. 137.



FIG. 140.



FIG. 145.



FIG. 148.



FIG. 144.

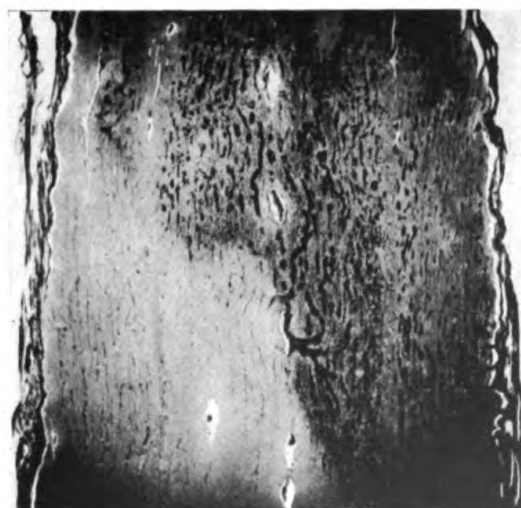


FIG. 147.



FIG. 143.



FIG. 146.



FIG. 151.

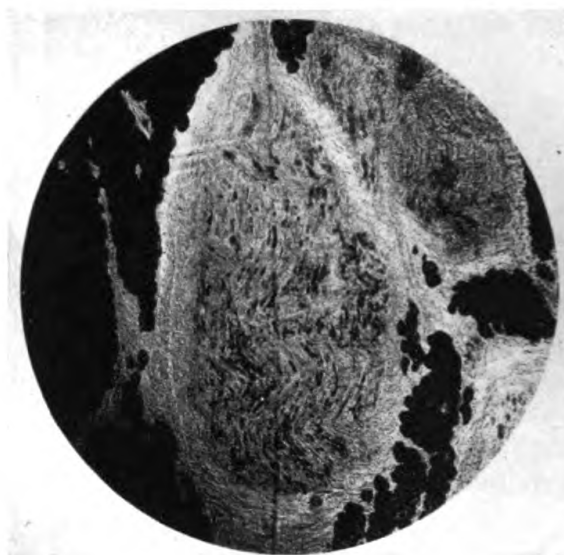


FIG. 154.



FIG. 150.

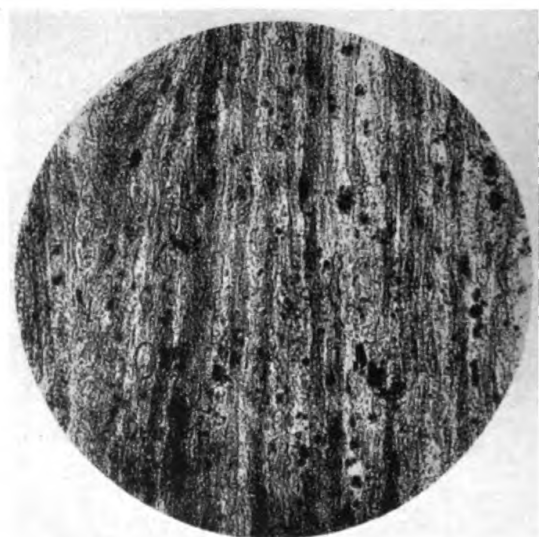


FIG. 153.

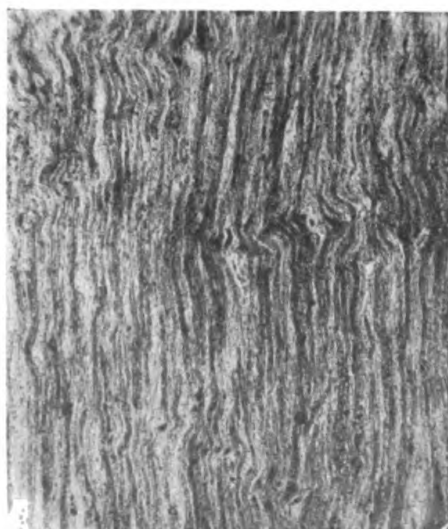


FIG. 149.

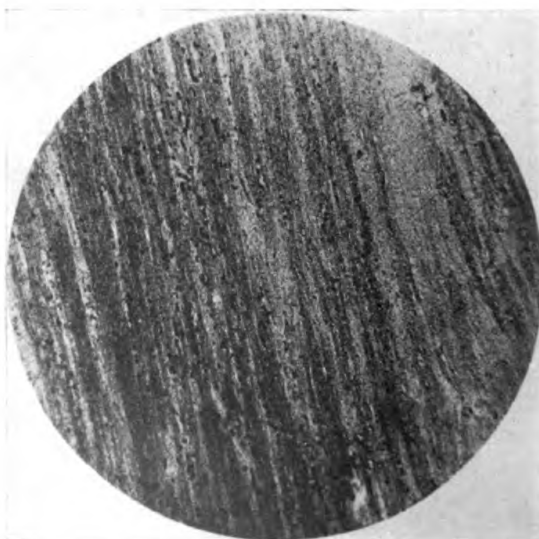


FIG. 152.

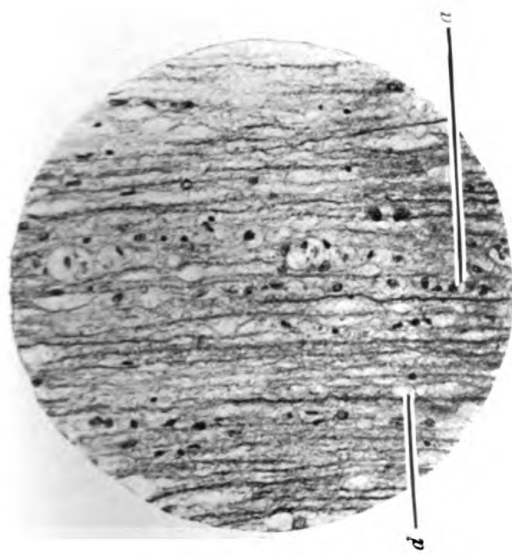


FIG. 155.

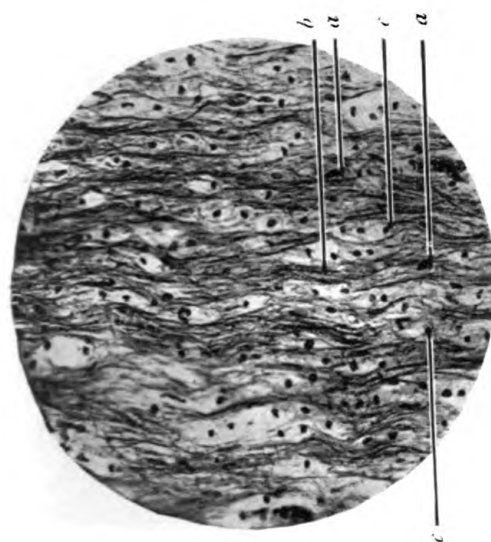


FIG. 156.

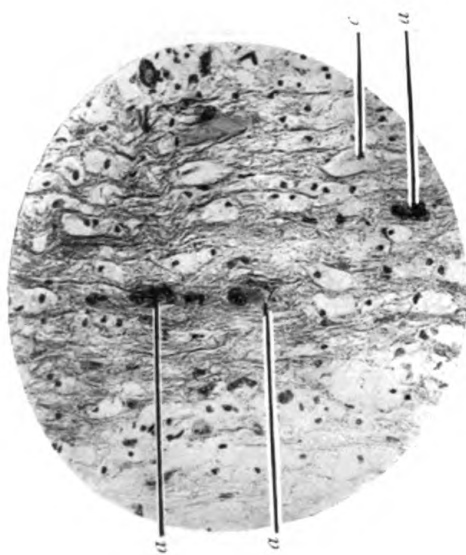


FIG. 157.

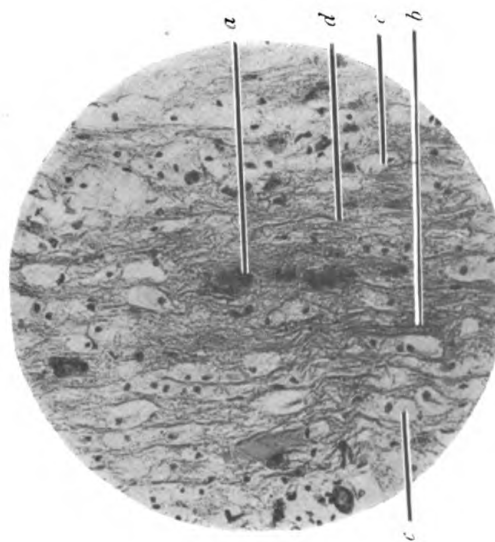


FIG. 158.

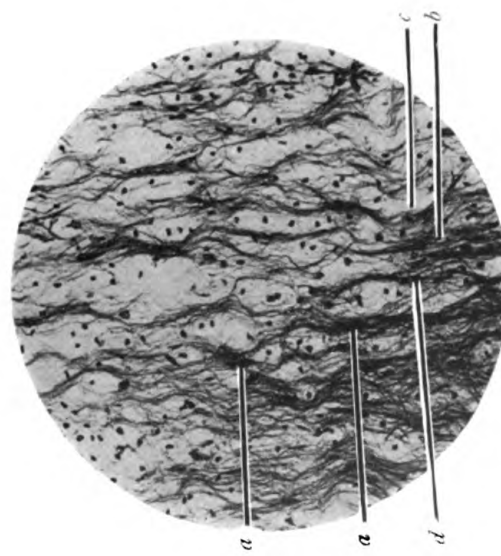


FIG. 159.

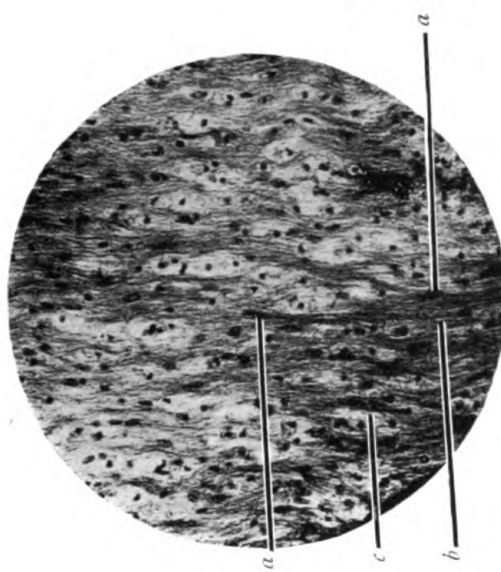


FIG. 160.

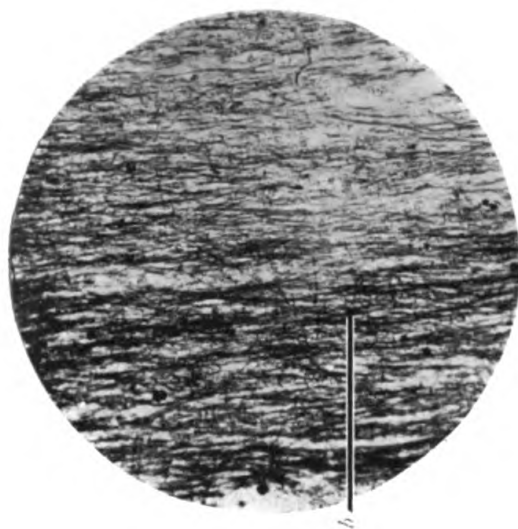


FIG. 163.

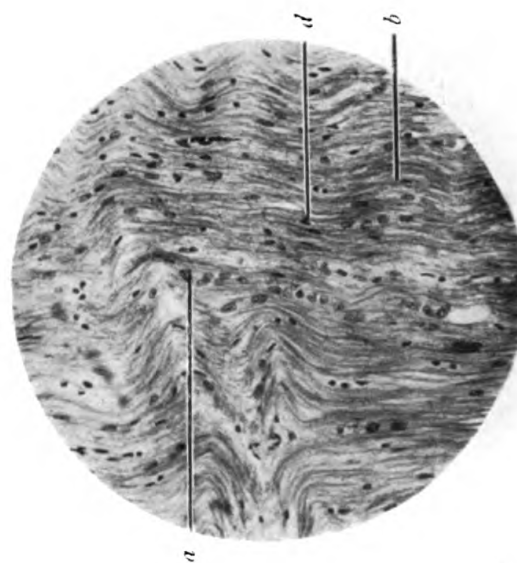


FIG. 166.

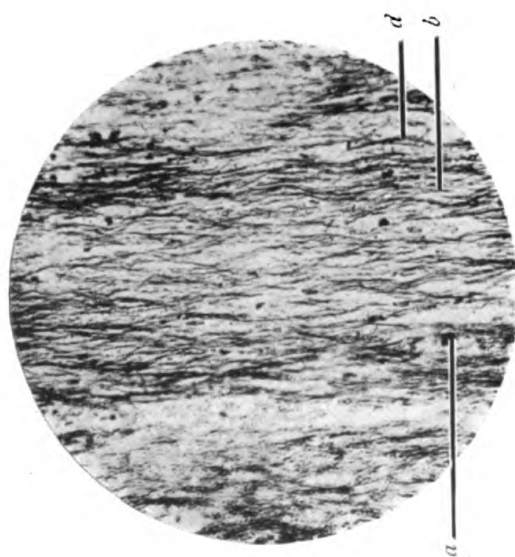


FIG. 162.

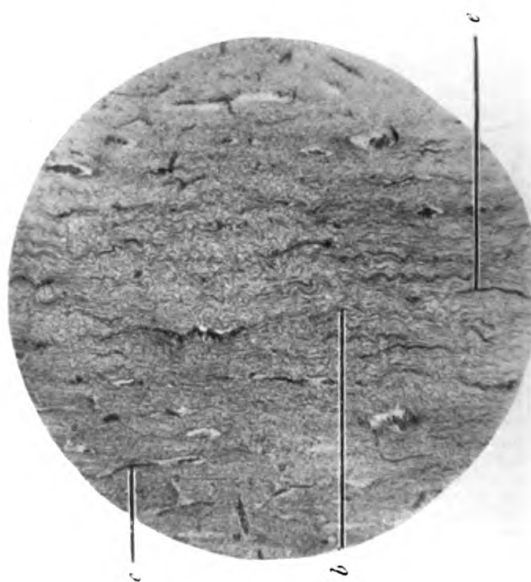


FIG. 165.

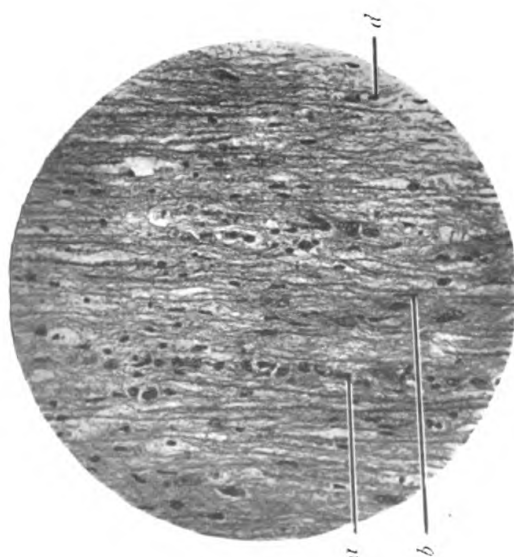


FIG. 161.

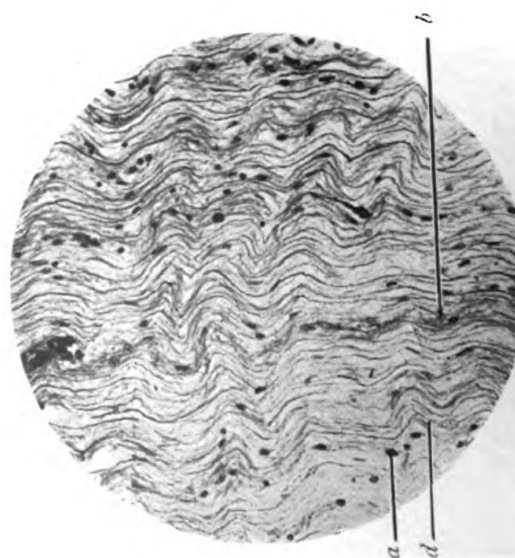


FIG. 164.

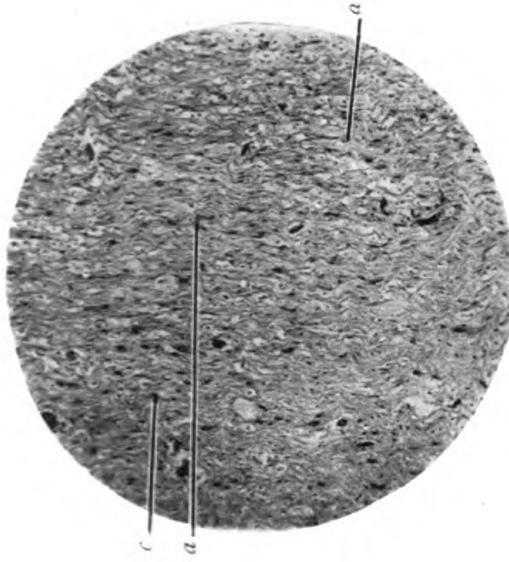


FIG. 169.

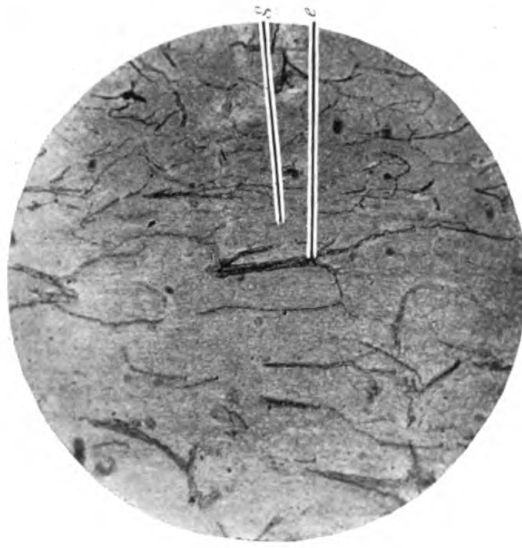


FIG. 172.

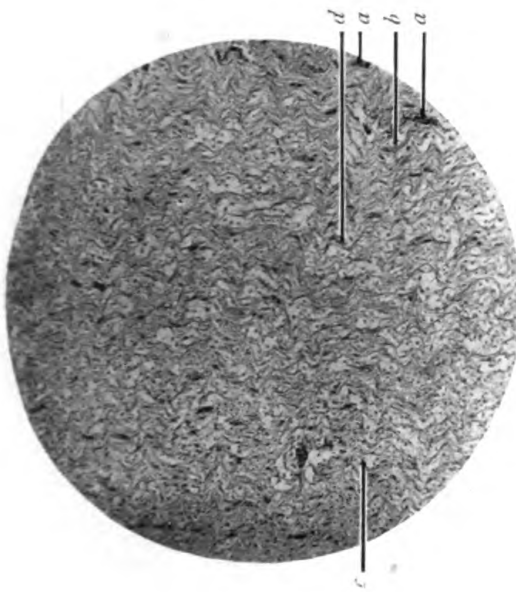


FIG. 168.

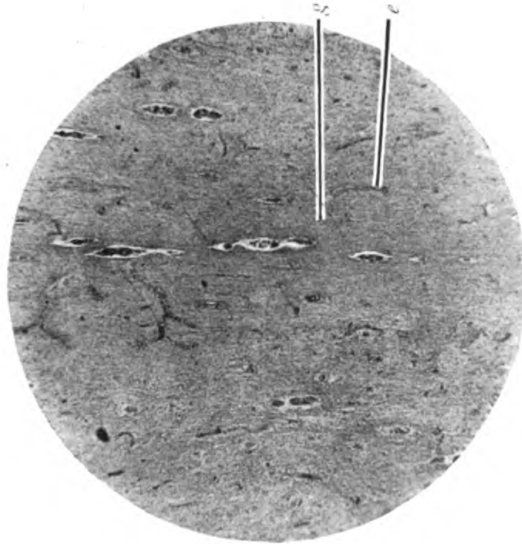


FIG. 171.

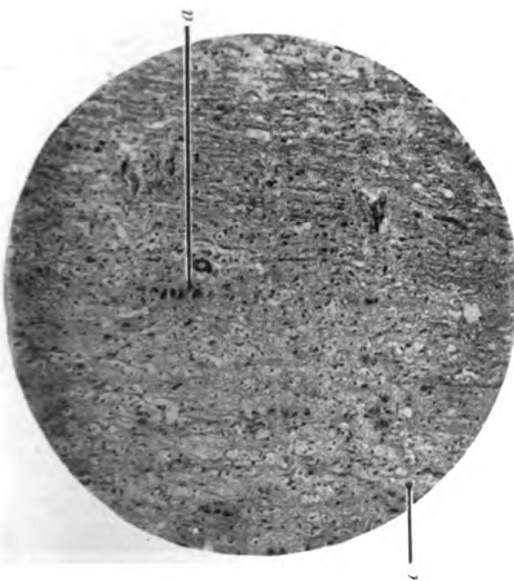


FIG. 167.

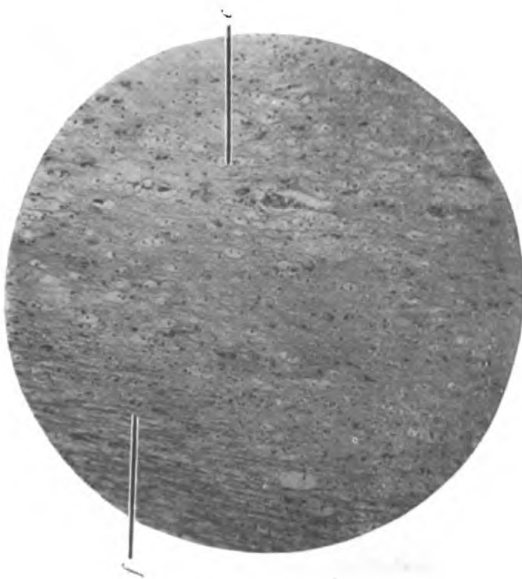


FIG. 170.

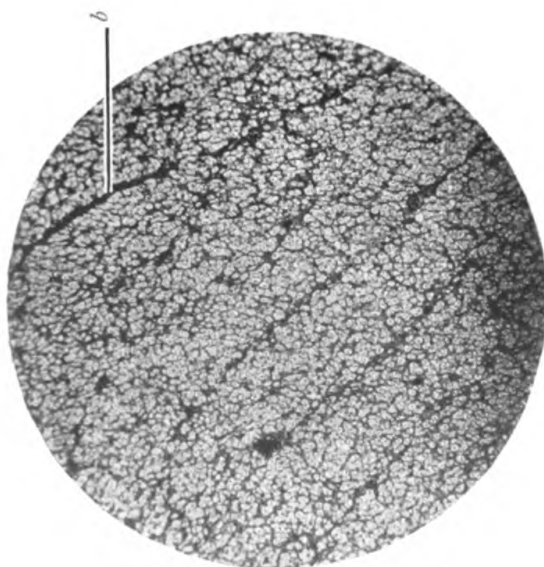


FIG. 173.

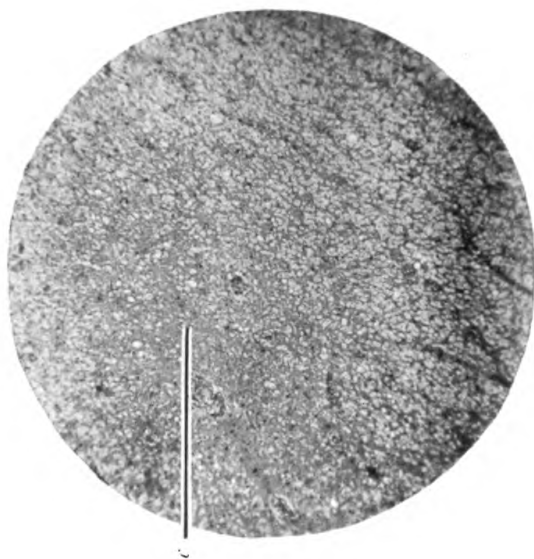


FIG. 174.

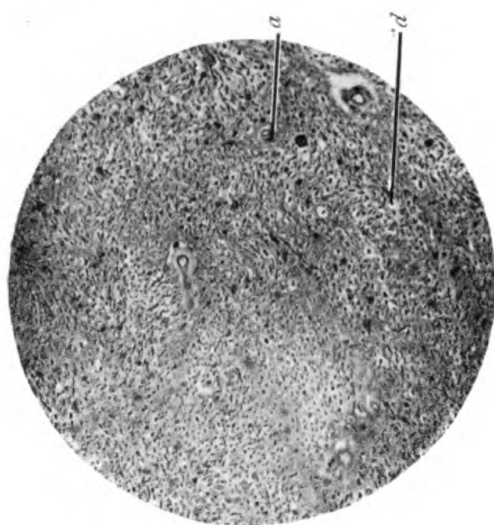


FIG. 175.

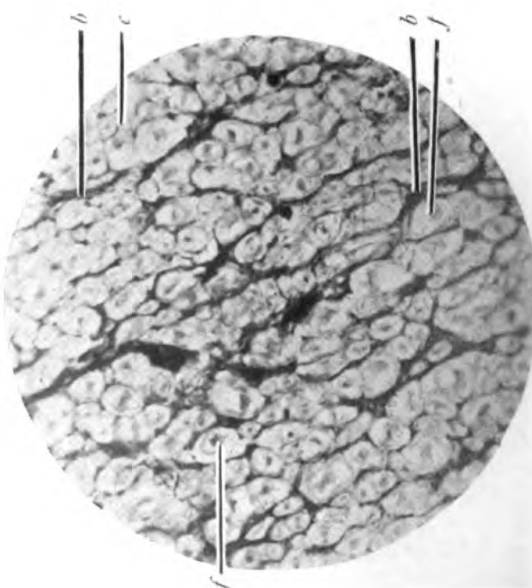


FIG. 176.

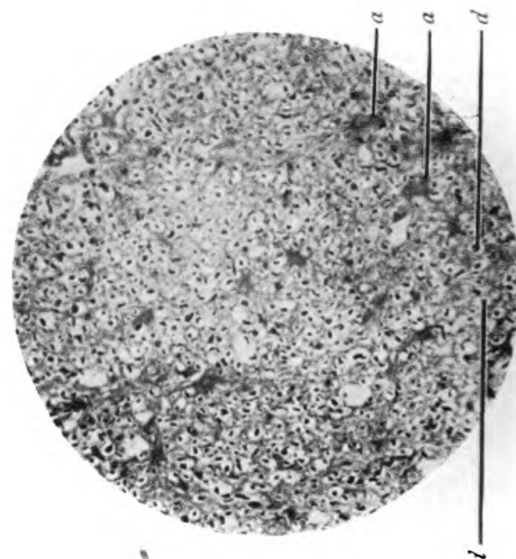


FIG. 177.

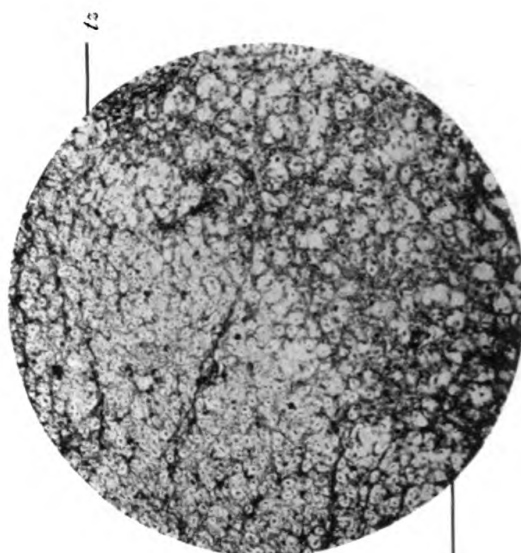


FIG. 178.

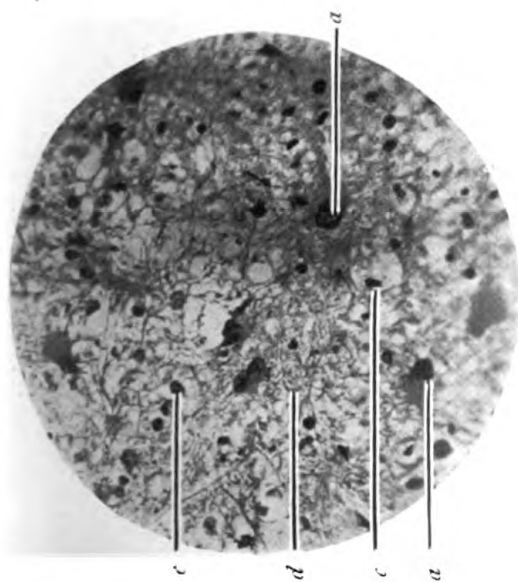


FIG. 179.

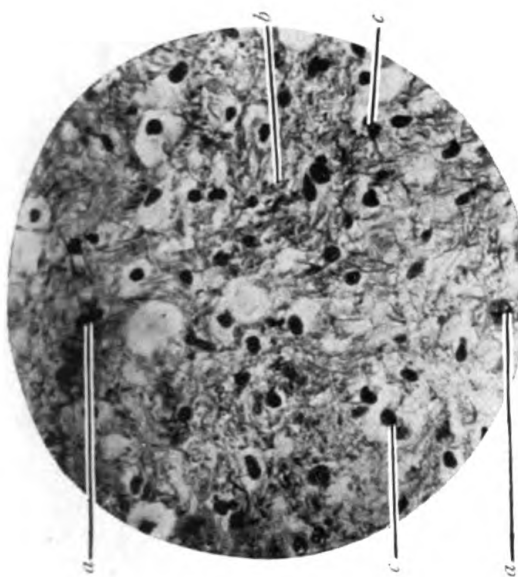


FIG. 180.

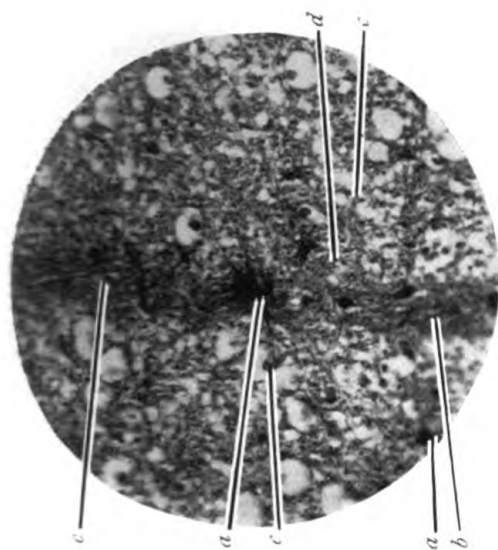


FIG. 181.

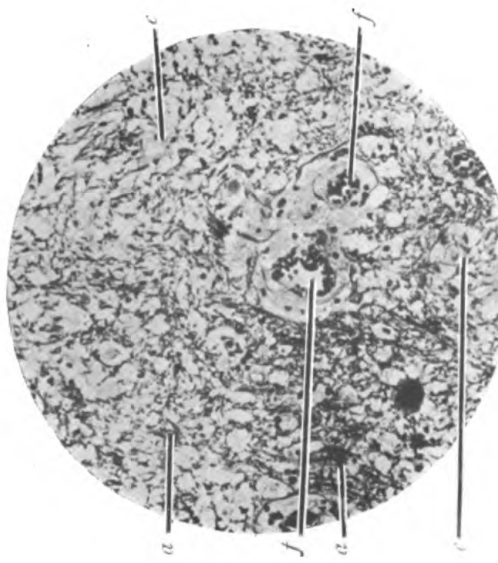


FIG. 182.

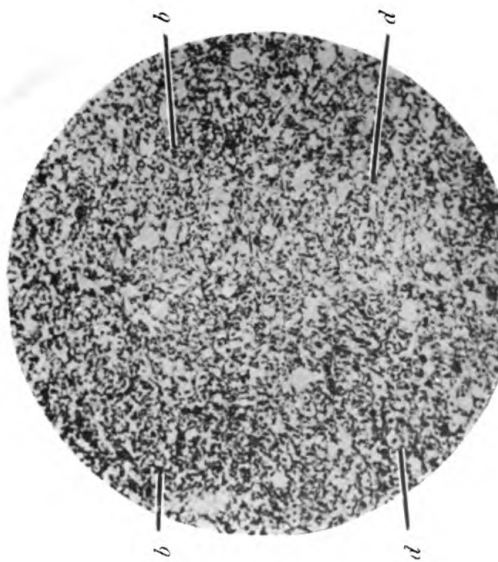


FIG. 183.

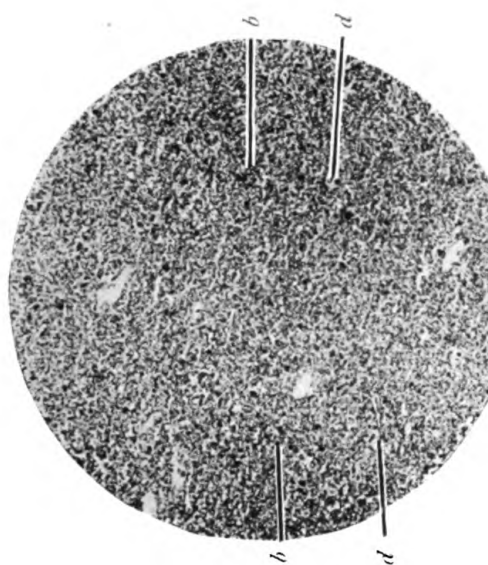


FIG. 184.

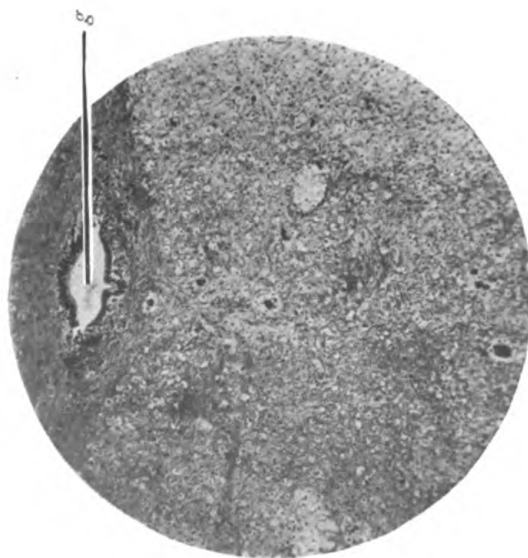


FIG. 185.

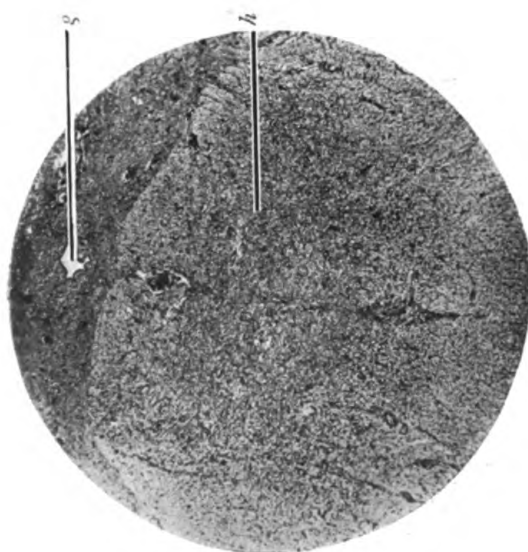


FIG. 186.

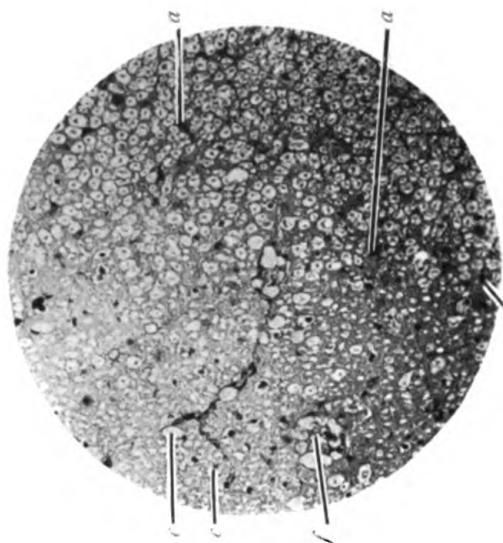


FIG. 187.

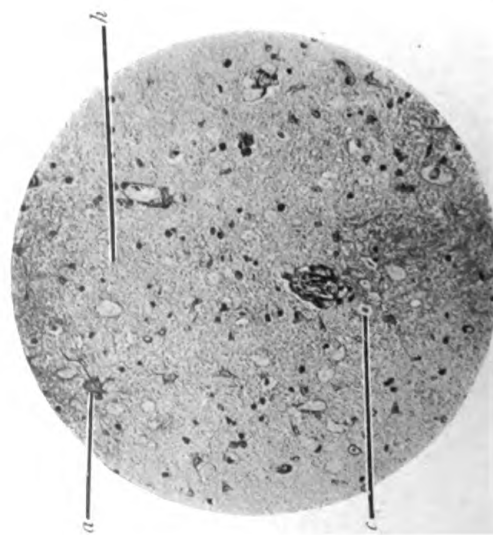


FIG. 188.

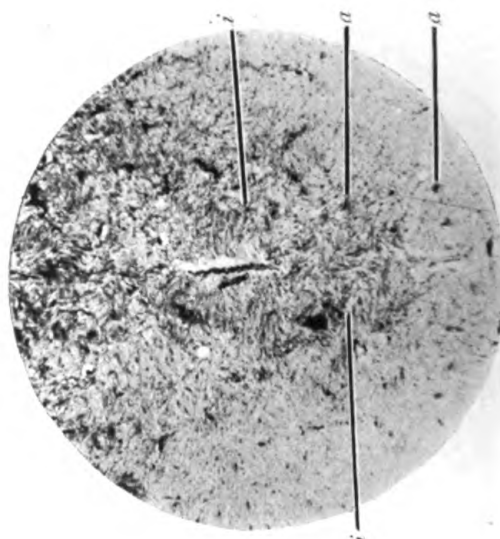


FIG. 189.

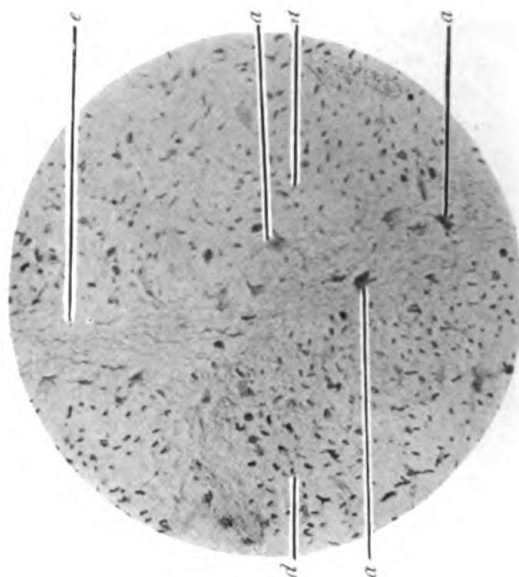


FIG. 190.

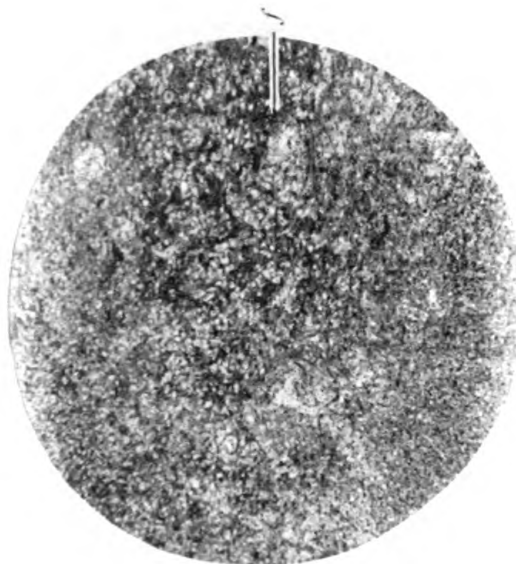


FIG. 193.

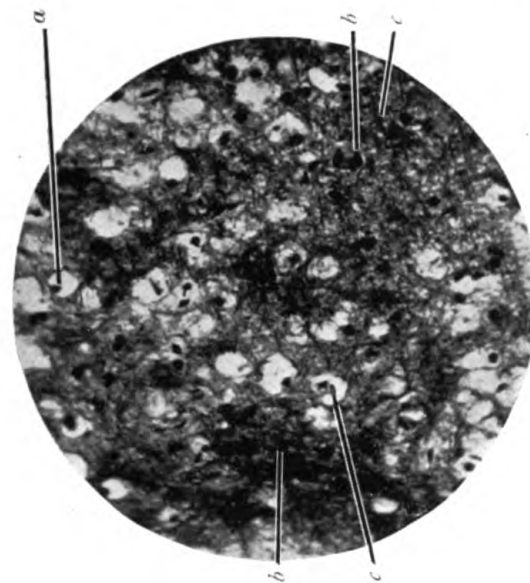


FIG. 196.

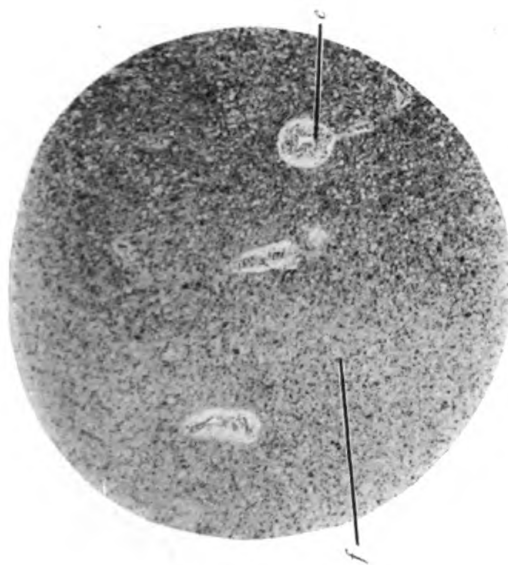


FIG. 192.

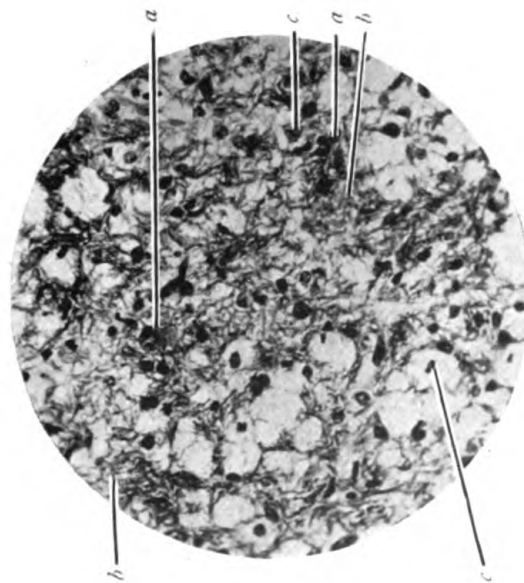


FIG. 195.

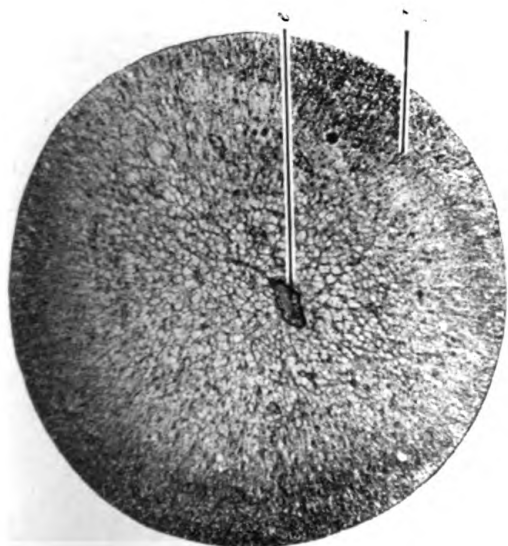


FIG. 191.



FIG. 194.

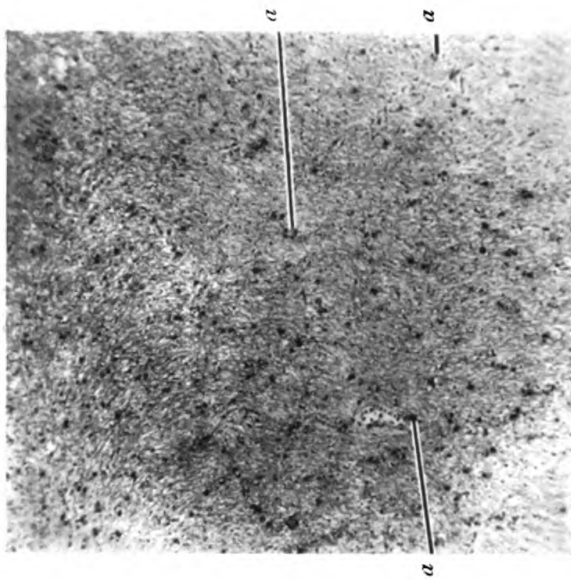


FIG. 199.

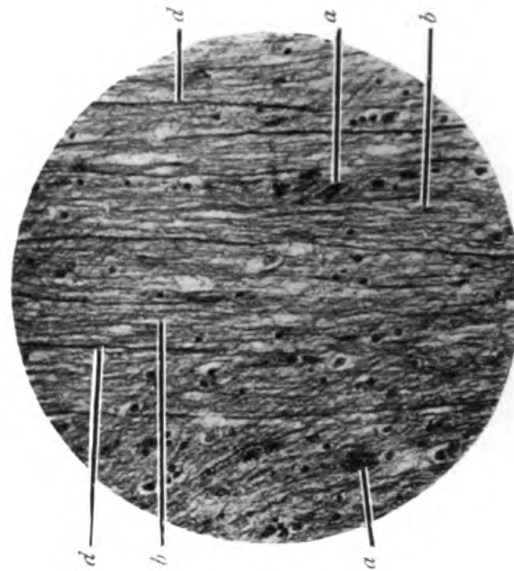


FIG. 202.

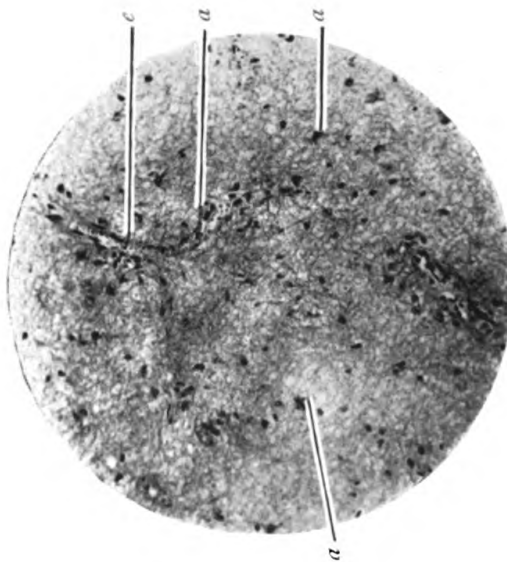


FIG. 198.

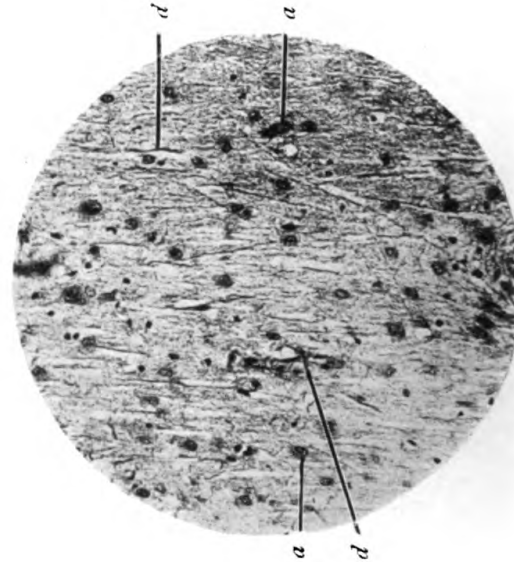


FIG. 201.

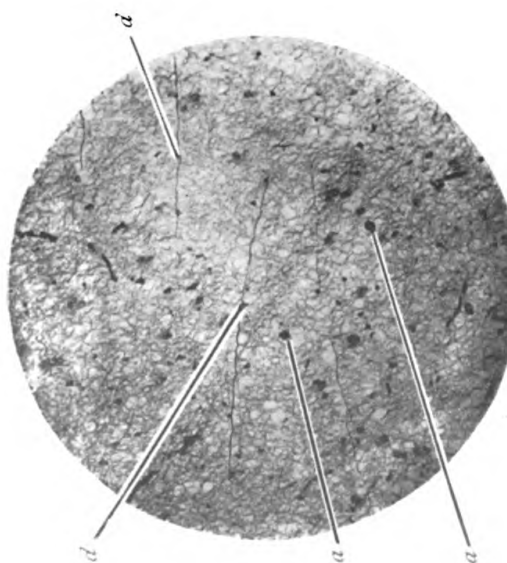


FIG. 197.



FIG. 200.

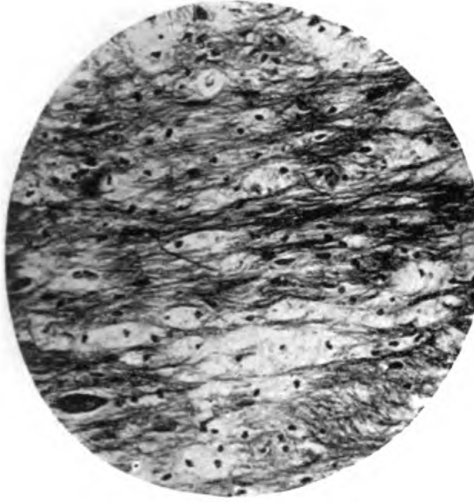


FIG. 205.

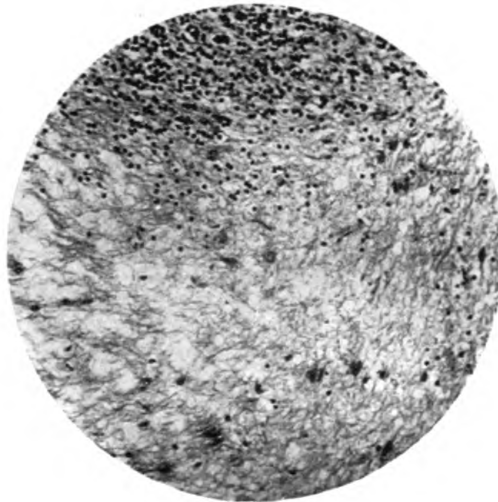


FIG. 208.



FIG. 204.

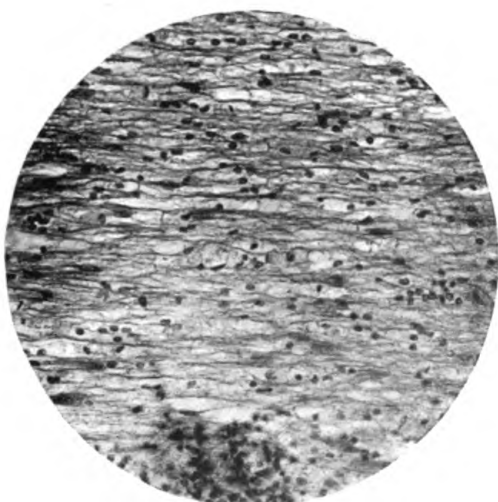


FIG. 207.



FIG. 203.

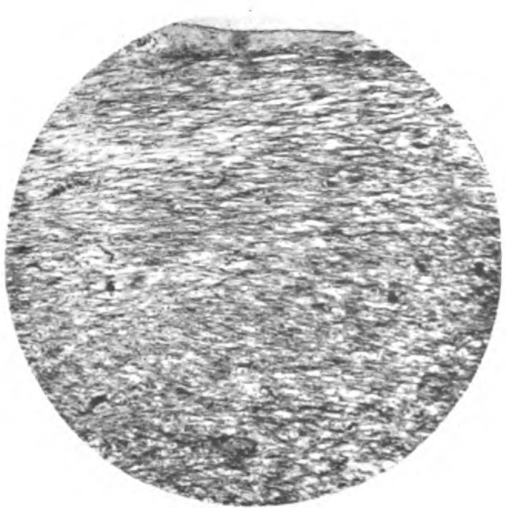


FIG. 206.

PLATE XXXVI.

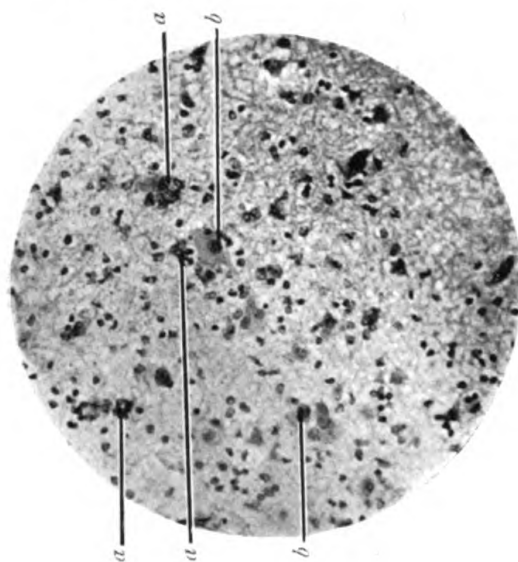


FIG. 211.

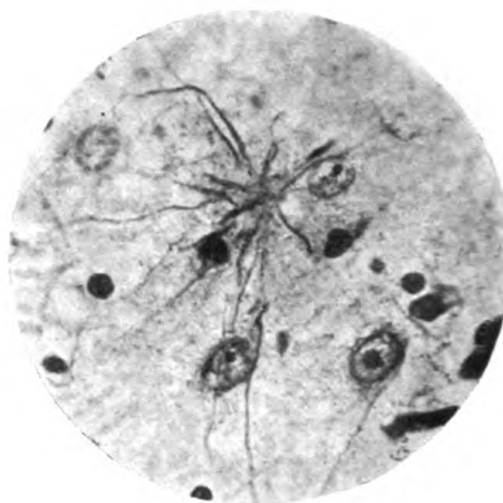


FIG. 214.

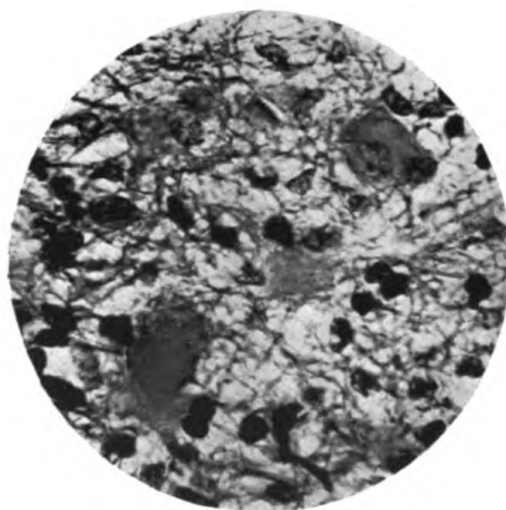


FIG. 210.

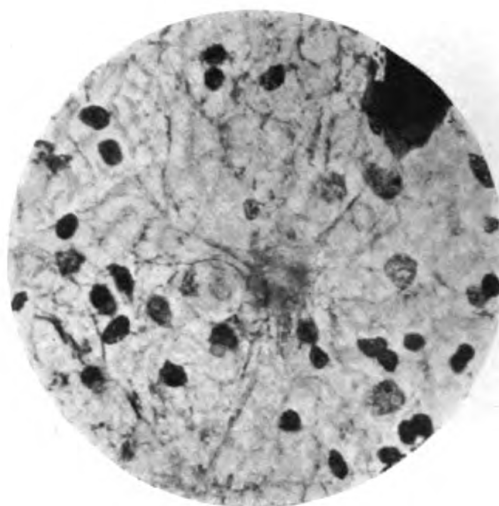


FIG. 213.

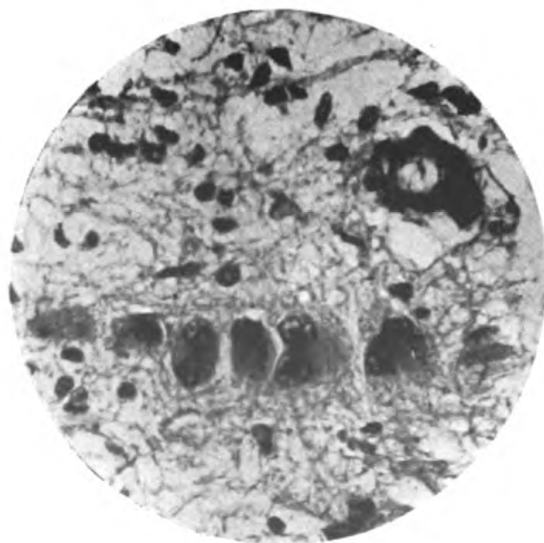


FIG. 209.

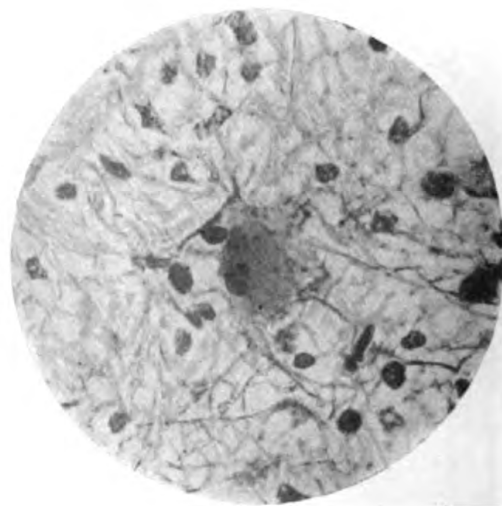


FIG. 212.

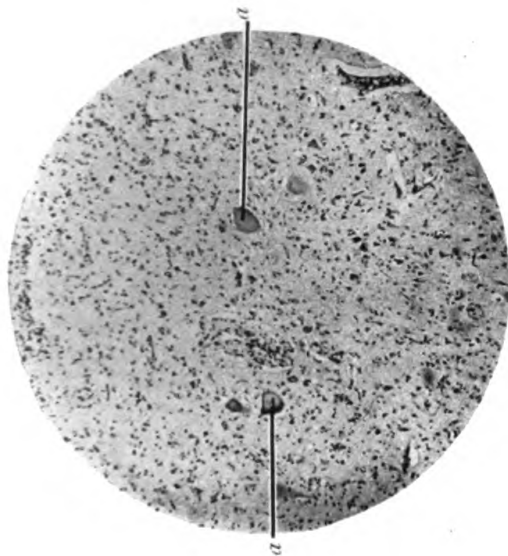


FIG. 217.

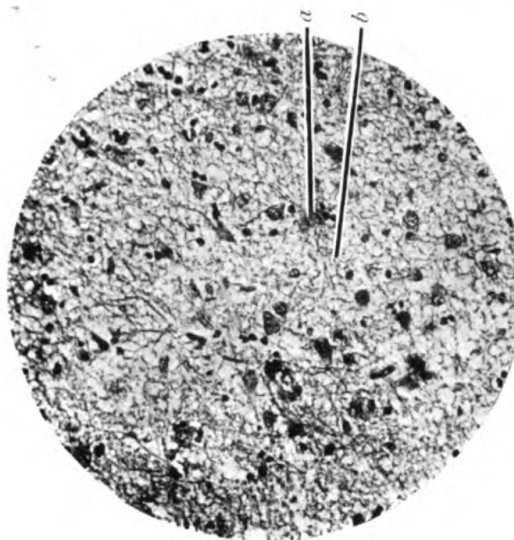


FIG. 220.

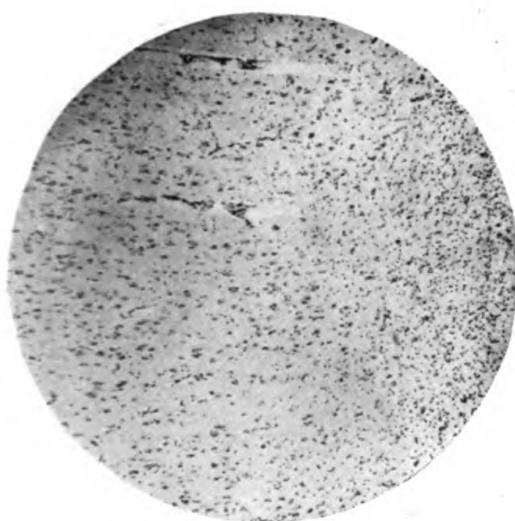


FIG. 216.

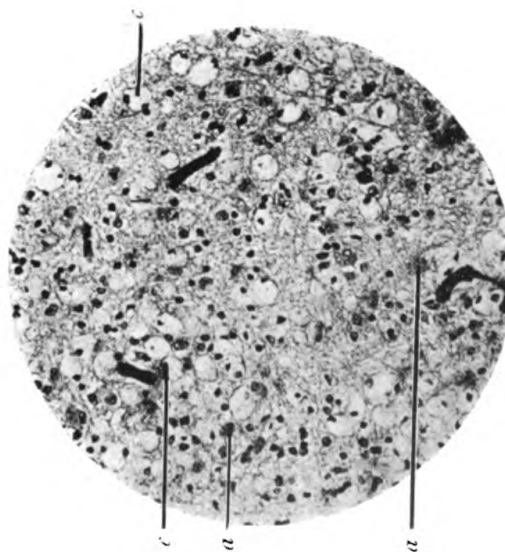


FIG. 219.



FIG. 215.

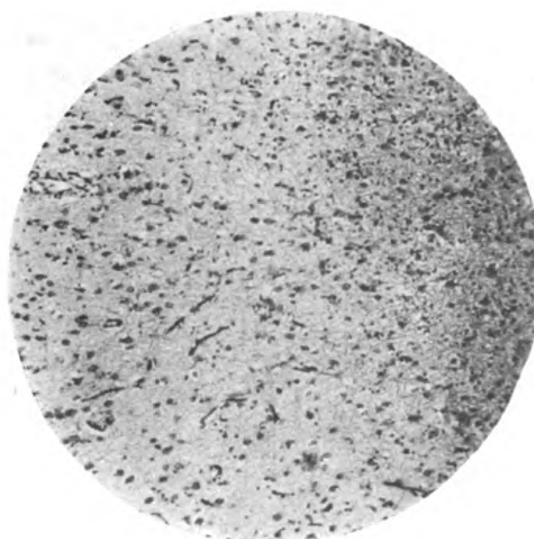


FIG. 218.

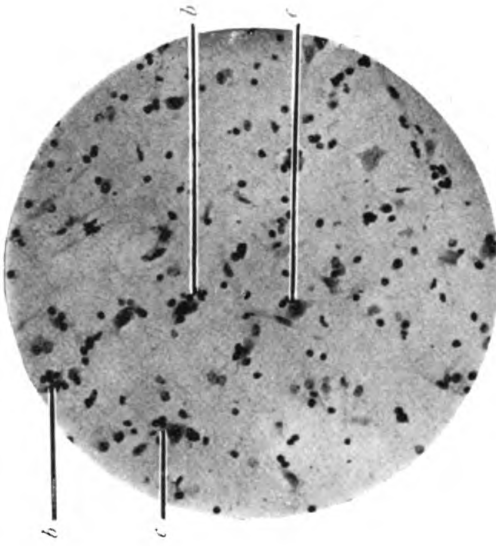


FIG. 223.

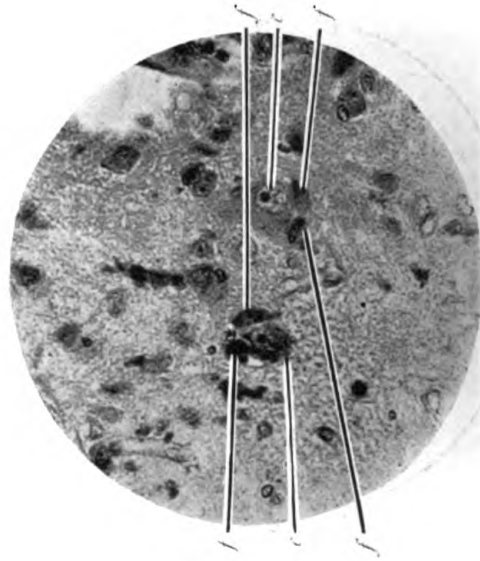


FIG. 226.

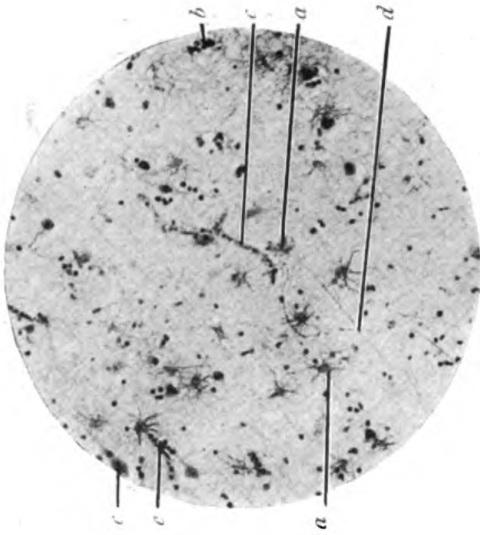


FIG. 222.

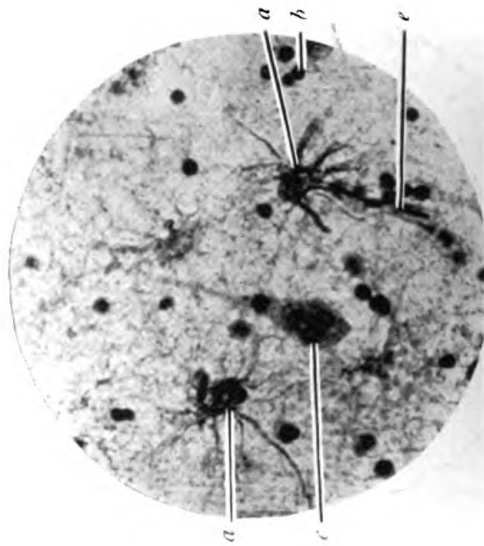


FIG. 225.

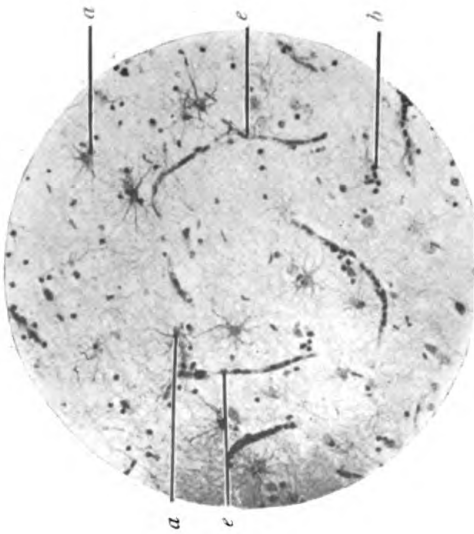


FIG. 221.

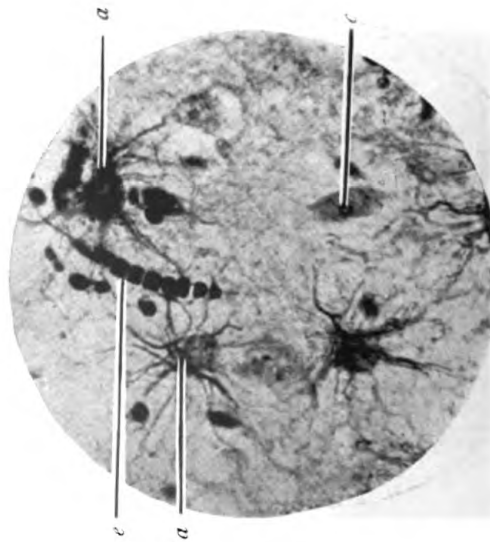


FIG. 224.



FIG. 229.

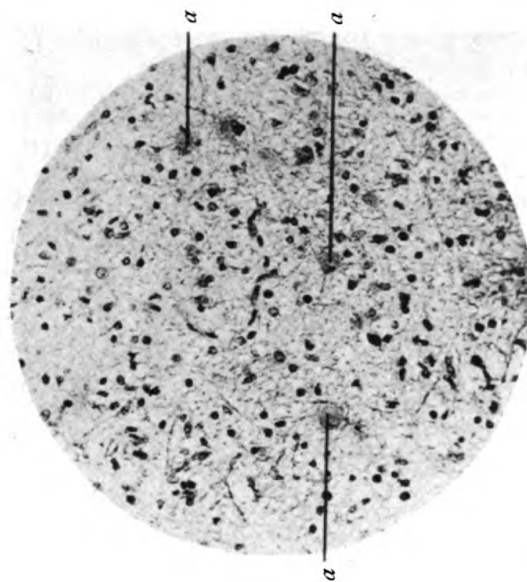


FIG. 232.

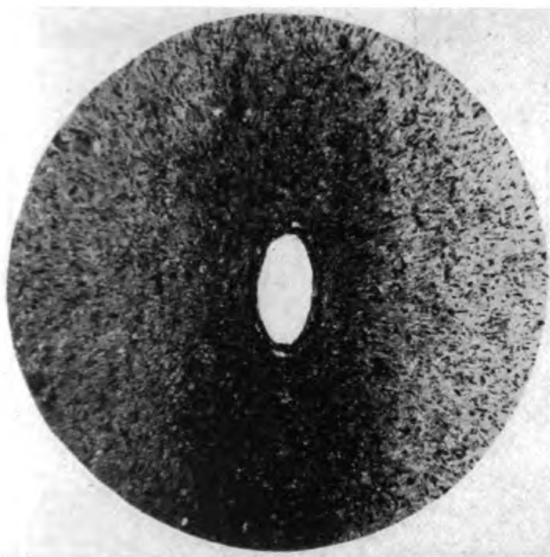


FIG. 228.

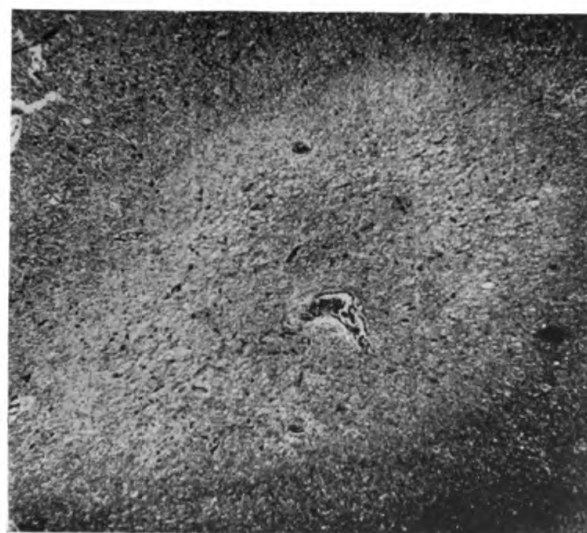


FIG. 231.



FIG. 227.

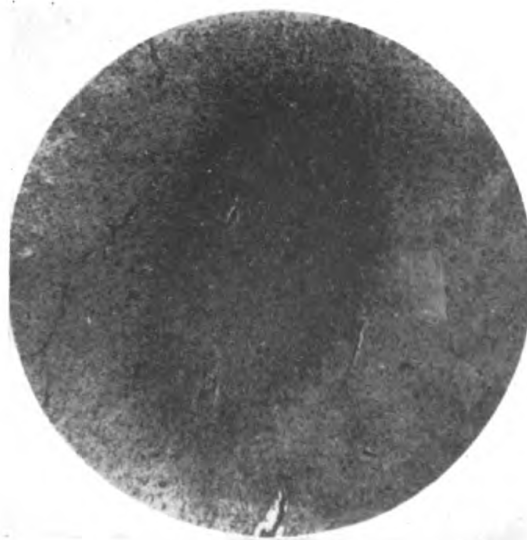


FIG. 230.

PLATE XL.

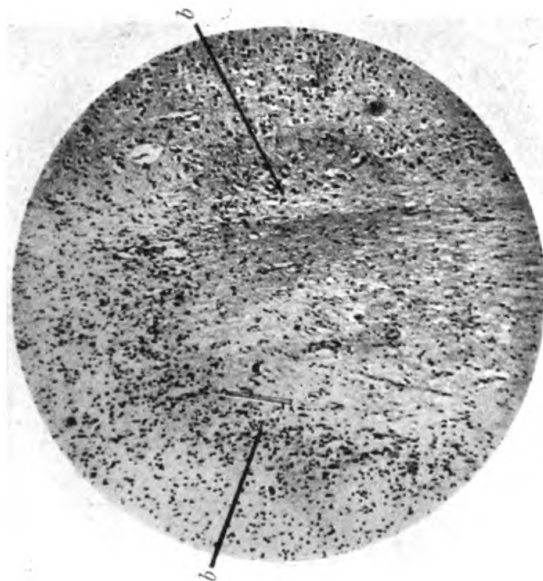


FIG. 235.

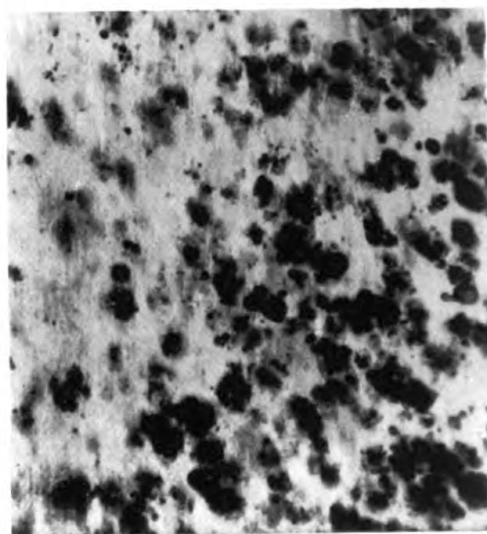


FIG. 238.

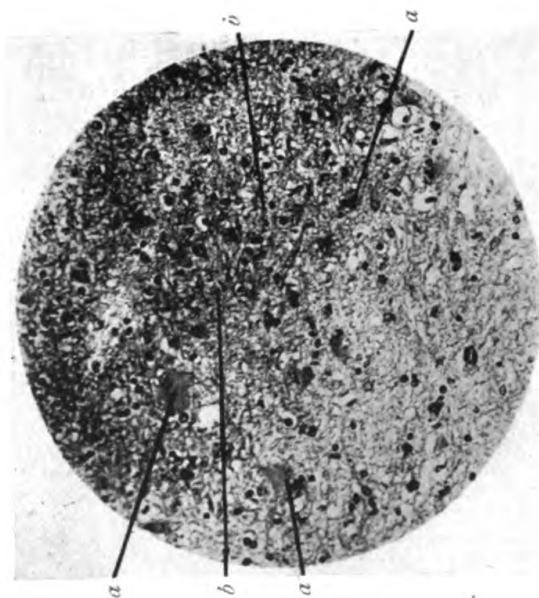


FIG. 234.



FIG. 237.

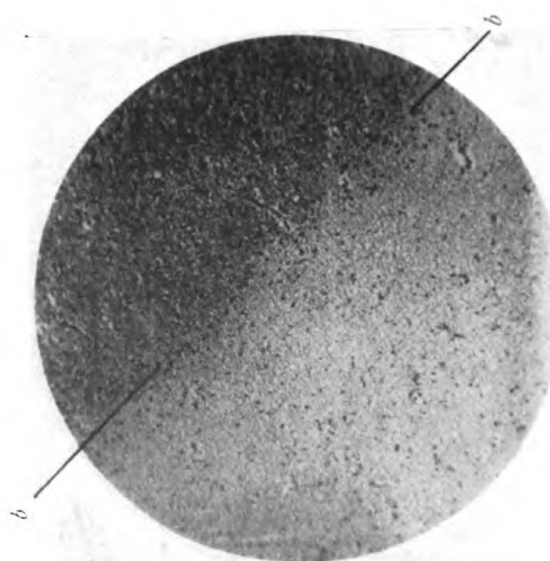


FIG. 233.



FIG. 236.

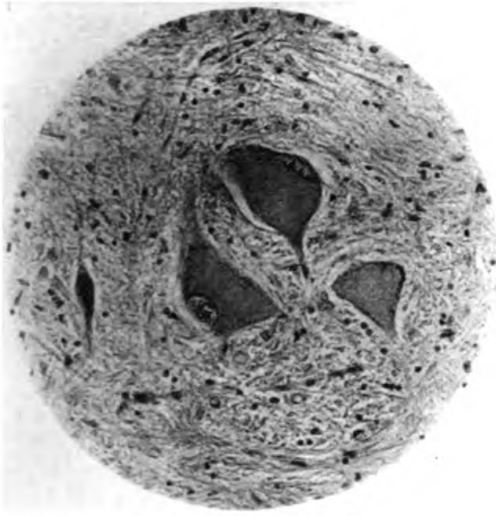


FIG. 241.

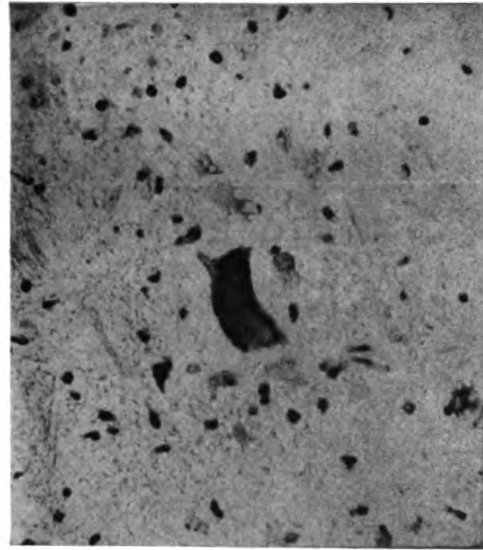


FIG. 244.

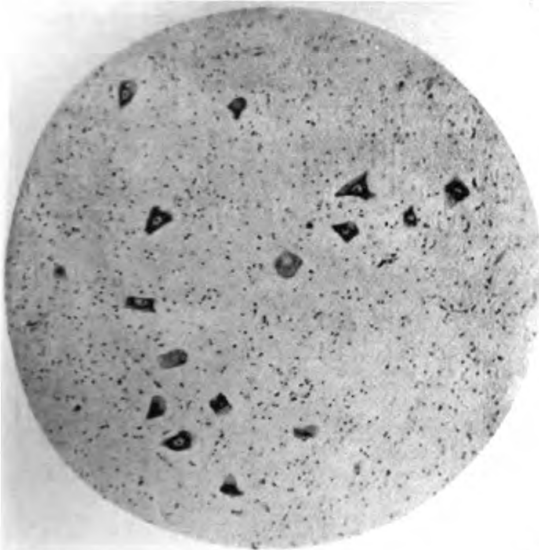


FIG. 240.

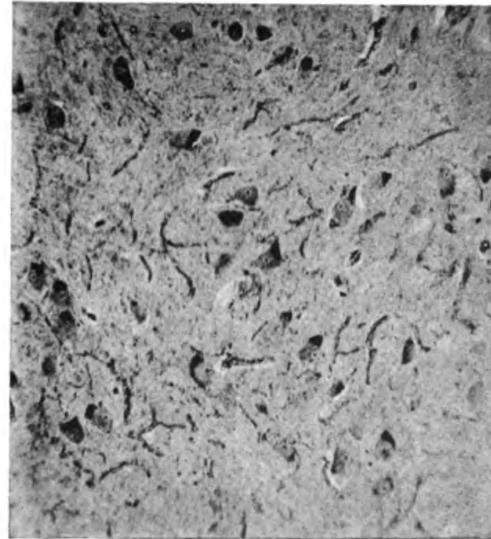


FIG. 243.

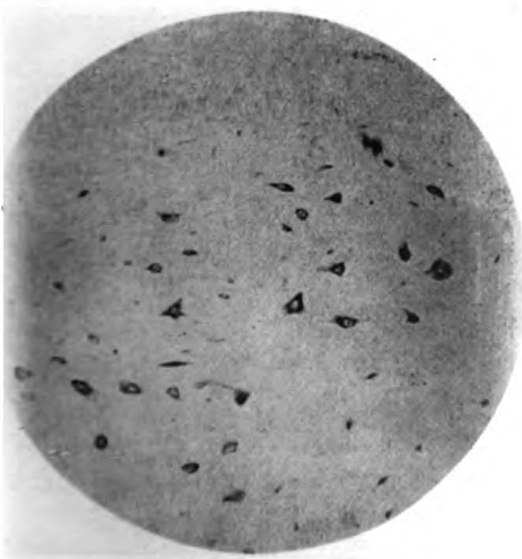


FIG. 239.

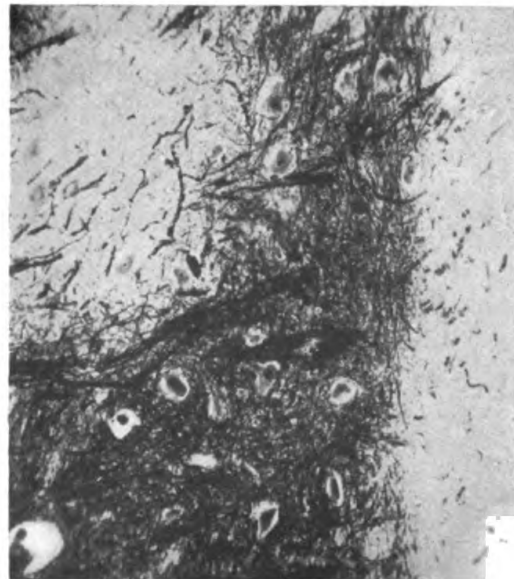


FIG. 242.



FIG. 247.

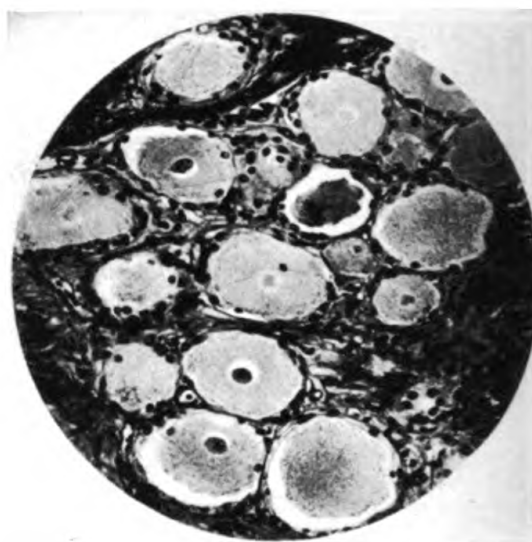


FIG. 250.

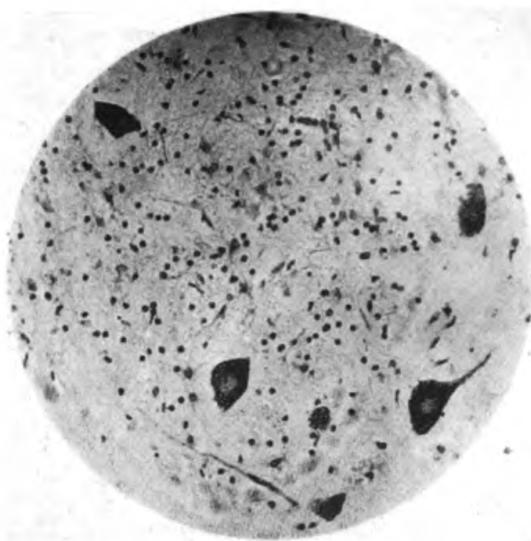


FIG. 246.

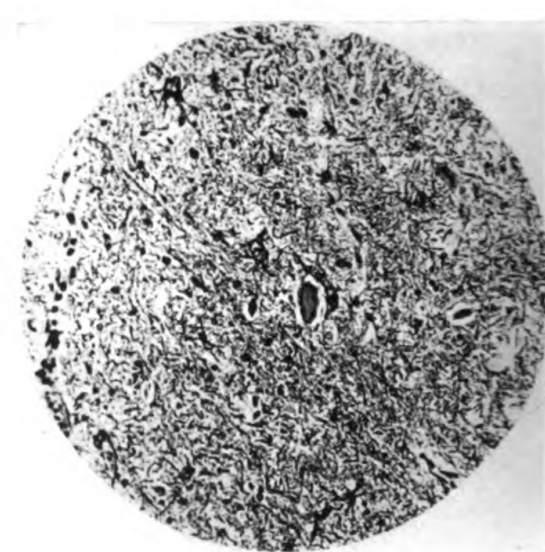


FIG. 249.

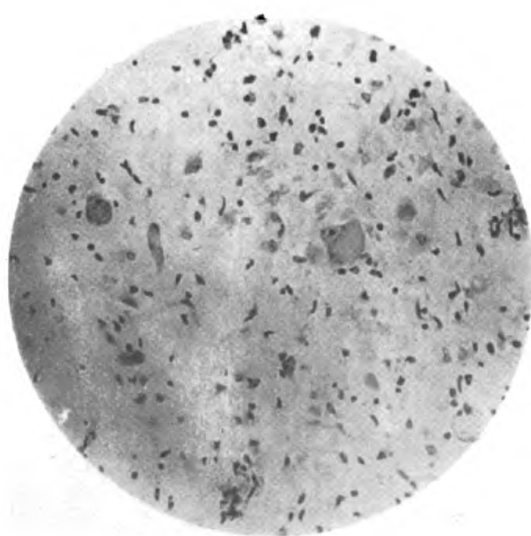


FIG. 245.



FIG. 248.

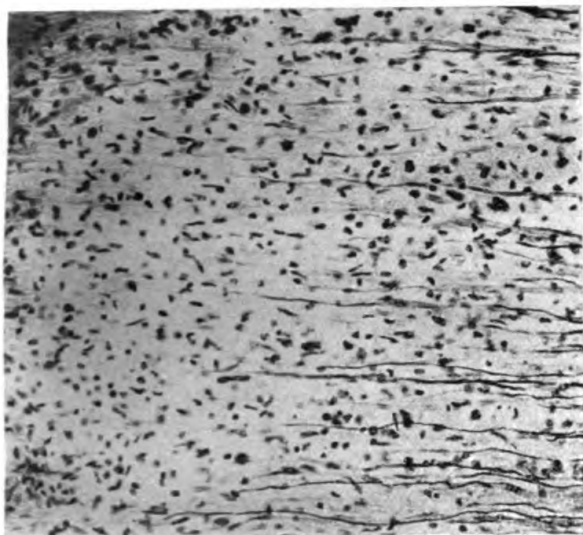


FIG. 253.

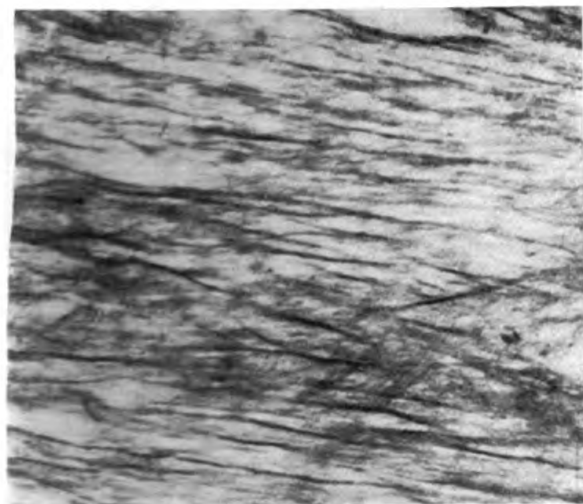


FIG. 252.



FIG. 251.



FIG. 256.

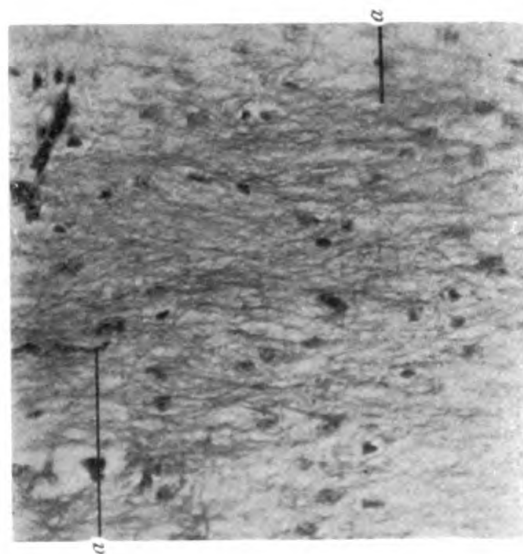


FIG. 255.



FIG. 254.

PLATE XLIV.



FIG. 259.

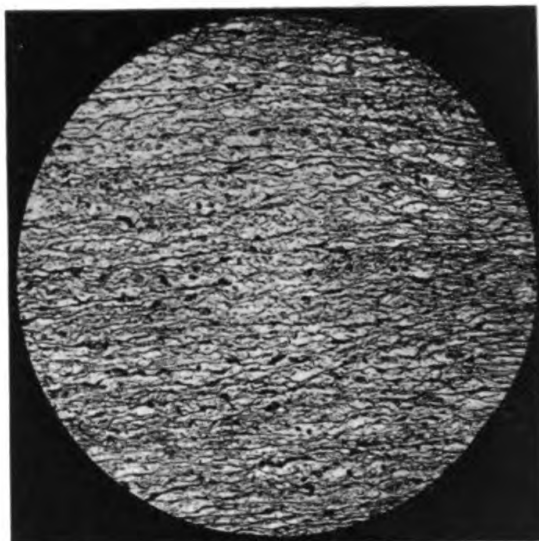


FIG. 262.

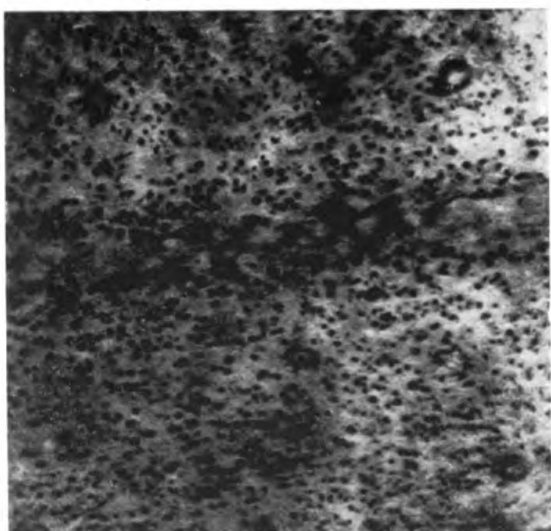


FIG. 258.

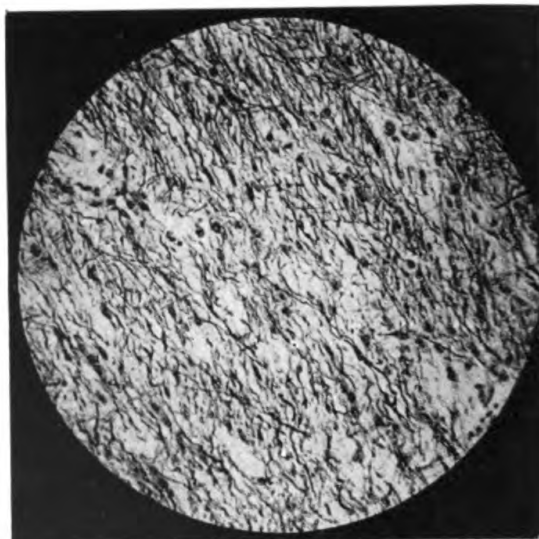


FIG. 261.

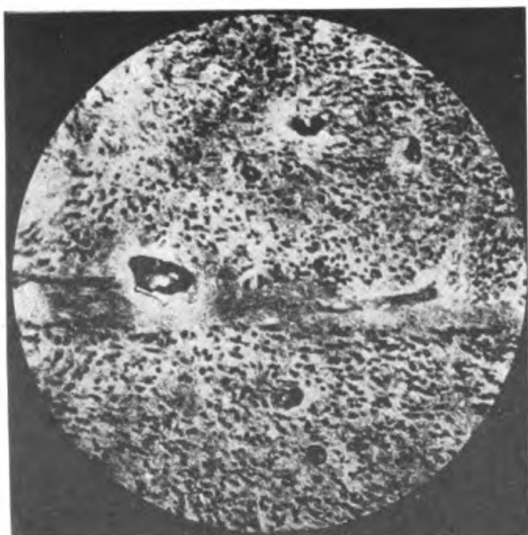


FIG. 257.

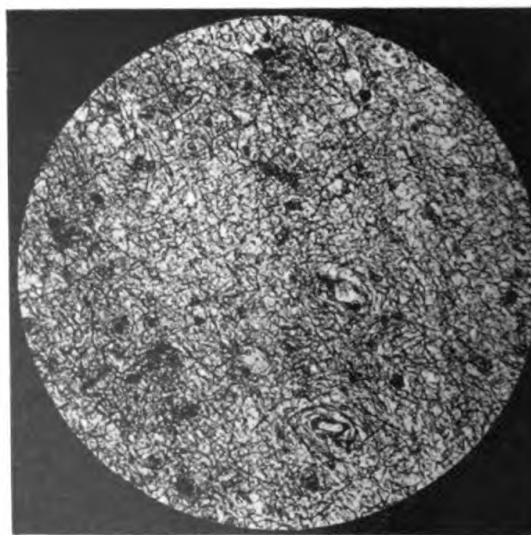


FIG. 260.

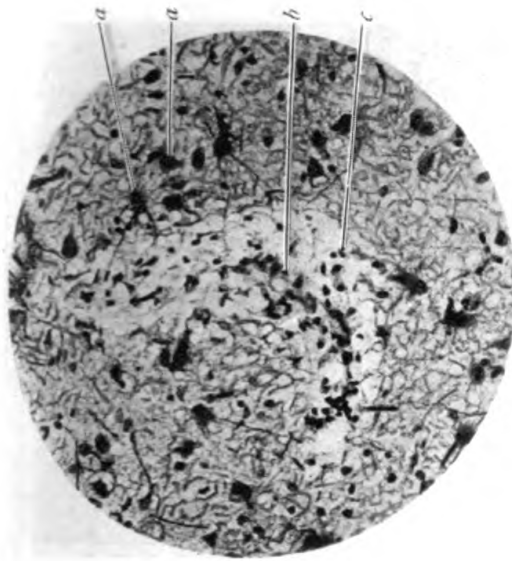


FIG. 265.

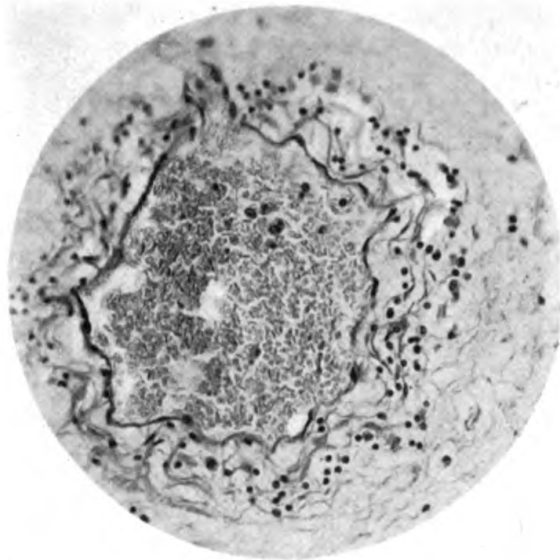


FIG. 268.

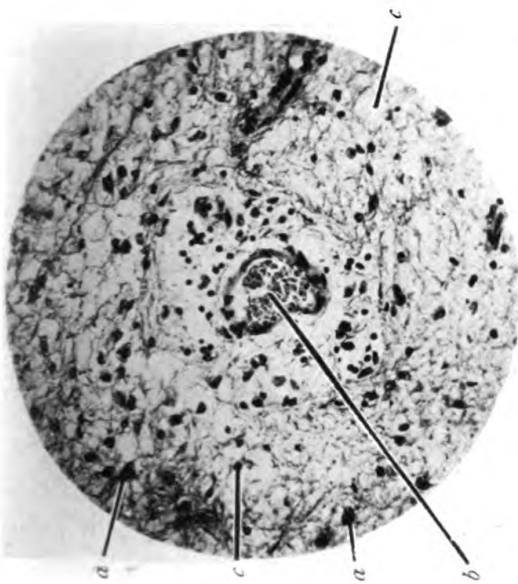


FIG. 264.

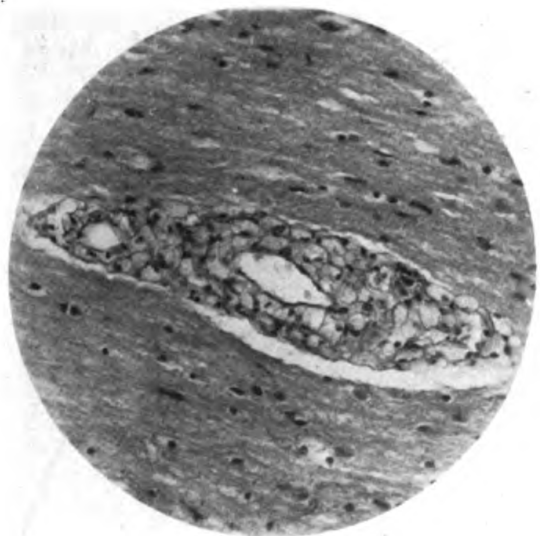


FIG. 267.



FIG. 263.

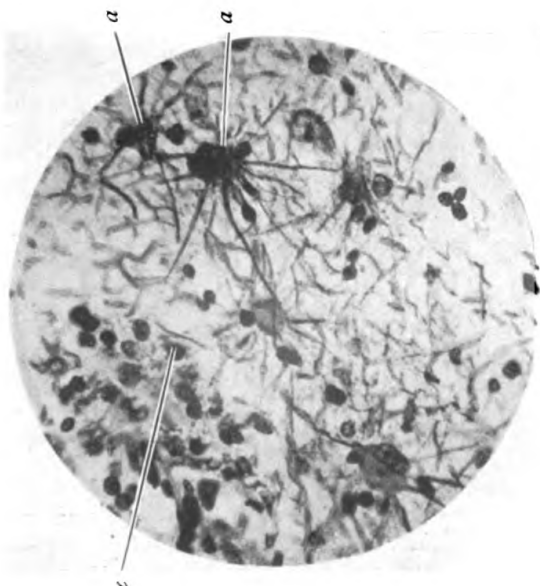


FIG. 266.

PLATE XLVI.



FIG. 271.



FIG. 274.

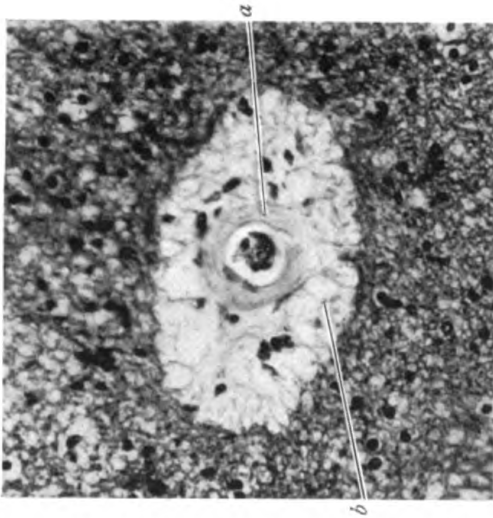


FIG. 270.

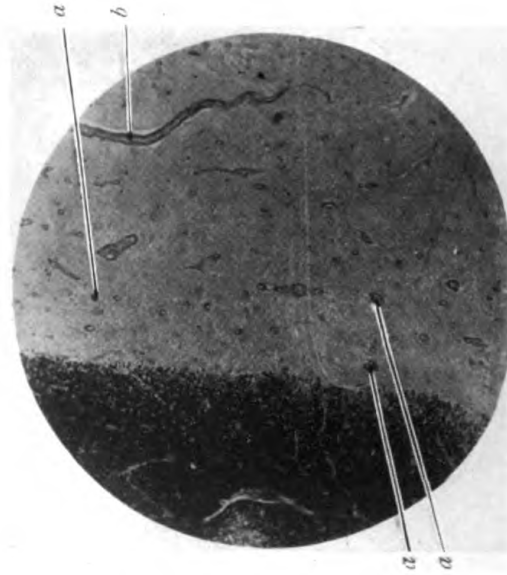


FIG. 273.

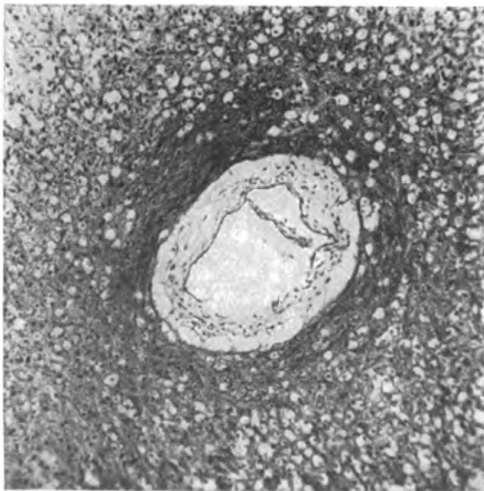


FIG. 269.

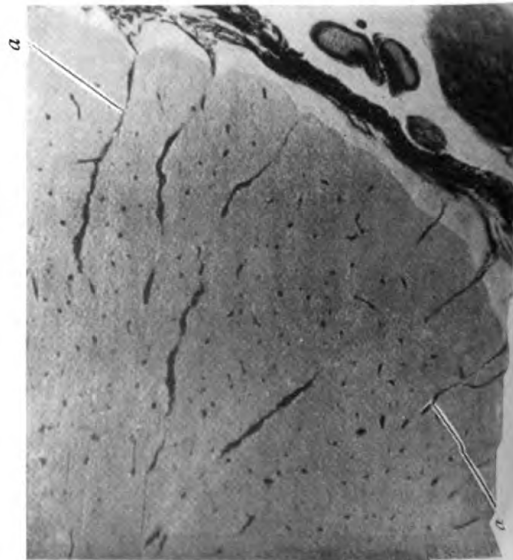


FIG. 272.

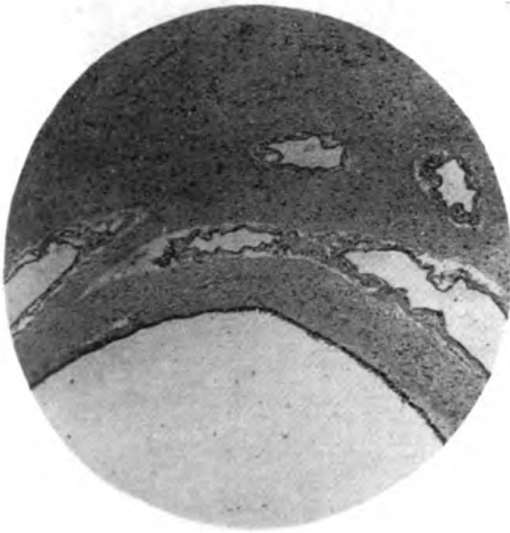


FIG. 277.



FIG. 280.

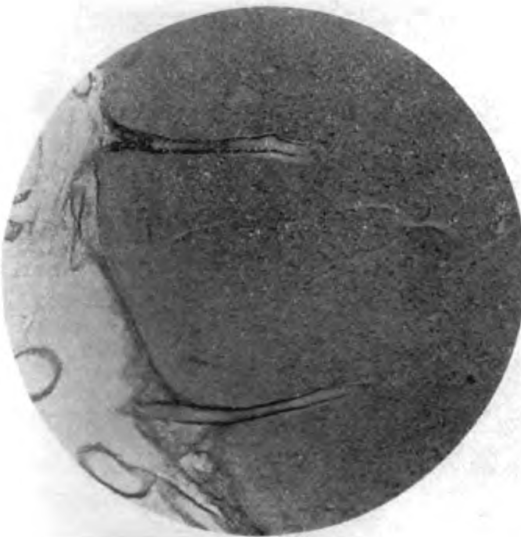


FIG. 276.

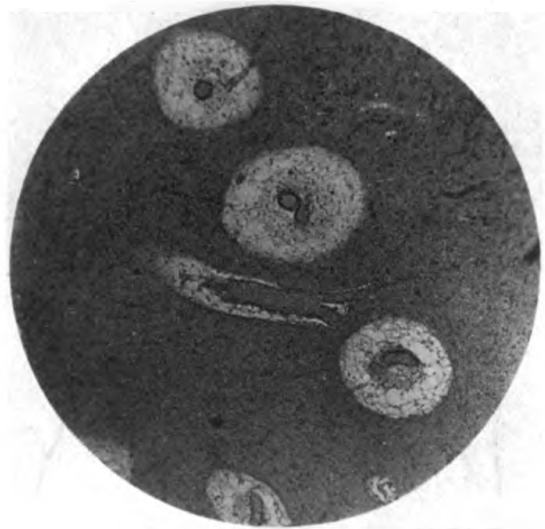


FIG. 279.



FIG. 275.

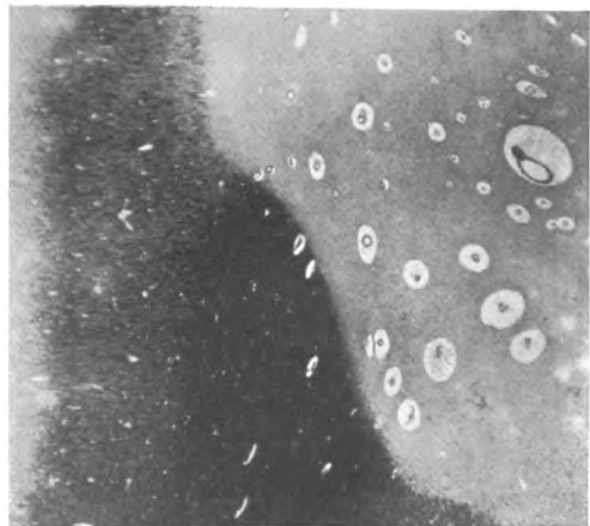


FIG. 278.



FIG. 283.

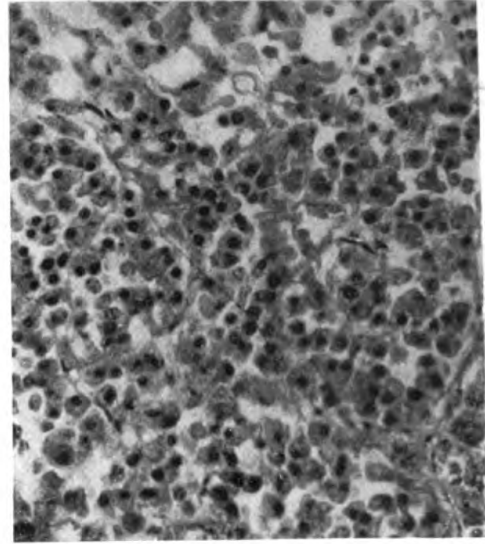


FIG. 286.

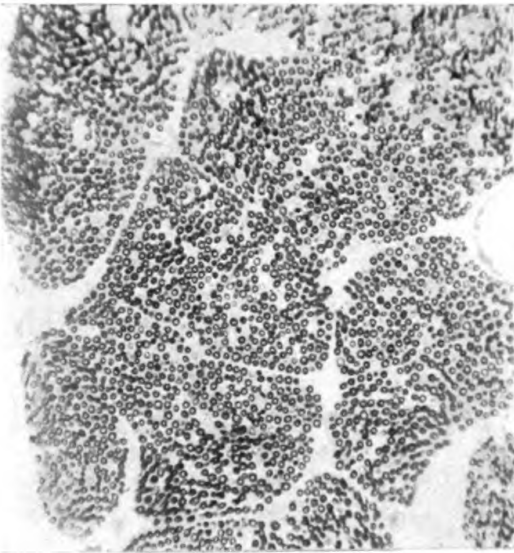


FIG. 282.

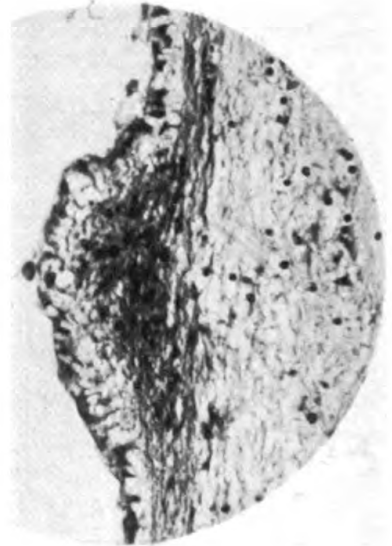


FIG. 285.

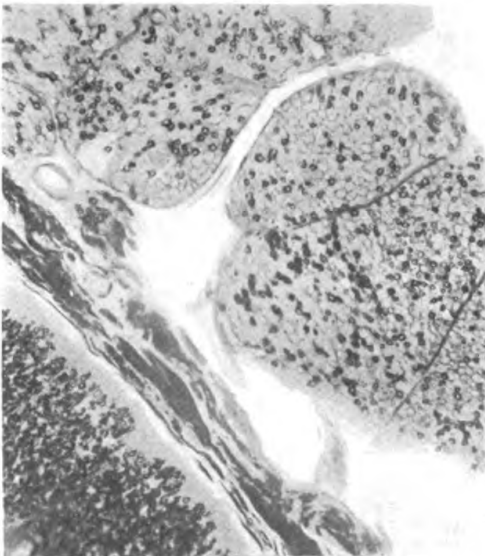


FIG. 281.

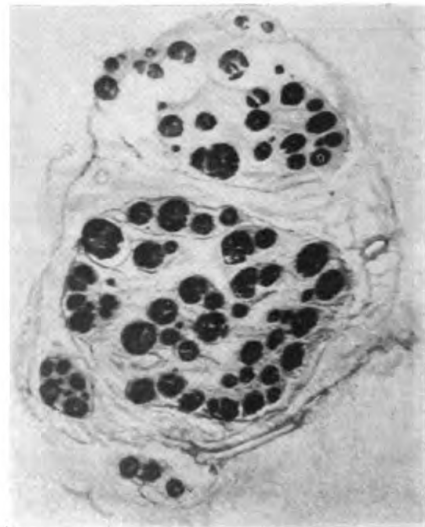


FIG. 284.

Review

of

Neurology and Psychiatry

Abstracts

ANATOMY.

NEOPALLIAL MORPHOLOGY OF FOSSIL MEN AS STUDIED
(69) **FROM ENDOCRANIAL CASTS.** M. BOULE and R. ANTHONY,
Journ. of Anat., 1917, li., Jan., p. 95.

THE authors consider that the results of Symington's researches (*v. Review*, 1916, xiv., p. 67), though of value for modern man, are not necessarily valid for the interpretation of a fossil Neanderthal man, where the shape of the cranium is so different. Therefore, it is not only permissible, but highly desirable, to endeavour in the case of this fossil man, as well as for any fossil Primate, to determine what positive information can be obtained from the endocranial cast, the only material available for such a purpose.

From the re-perusal of their papers relating to the endocranial casts of the La Chapelle aux Saints, La Quina, and Neanderthal crania, it seems clear to them that they sufficiently emphasised the fact of the hypothetical character of some of their suggestions in reference to neopallial morphology, that they laid special stress upon the lack of clear indications of the neopallial features upon the upper surface of the cast, as is the case in most human and anthropoid specimens, and that they refrained from assuming the presence of features of which no trace could be detected. Hence they think Symington's arguments and criticism are wholly irrelevant.

A. NINIAN BRUCE.

STUDIES ON THE OLFACTORY BULBS OF THE ALBINO RAT.—
(70) **I. Effect of a defective diet and of exercise. II. Number of cells in bulb.** CAROLINE M. HOLT, *Journ. Comp. Neurol.*, 1917, xxvii., Feb., pp. 201-252. (4 plates.)

THE regions anterior to the cerebrum, which are commonly considered the bulbs, are not strictly homologous for bulbs of different

ages and sizes, since in the brains of young or stunted rats a larger proportion of the bulb lies beneath the cerebrum than in better developed brains.

All layers of the olfactory bulb are about equally concerned in the increase in size or in the arrest of development of the bulb. The small cells of the molecular layer show a larger amount of cytoplasm in large bulbs than in small ones. The number of small cells in the molecular layer, apparently, is not correlated either with the age of the rat or size of the bulb. The entire computed number for a small, medium, and large bulb was found to be approximately 1,000,000 cells \pm 2 per cent. The mitral cells of small bulbs are smaller, on the average, than those of large bulbs. Within the limits here taken, the number of mitral cells is not affected by the age or the size of the bulb. There seems to be some variation between litters in the numbers of the mitral cells. The average number for thirteen bulbs was 76,650, the lowest number being 70,625, and the highest 83,974. The standard deviation σ is 4,564, and the probable error of the mean \pm 855.

When members of the same litter are compared, bulbs stunted by a defective diet or enlarged by exercise show practically the same number of mitral cells as do their controls. The mean difference is -1.8 per cent. for the tests.

The olfactory bulb size is correlated with cell size and not with cell number.

A. NINIAN BRUCE.

**SOME OBSERVATIONS UPON THE ANATOMICAL RELATIONS
(71) OF THE OPTIC NERVES AND CHIASMA TO THE
SPHENOID BONE.** G. F. WALLIS, *Practitioner*, 1917, xcvi.,
pp. 41-52. (Illust.)

THE author records observations on ten bodies with regard to the position of the optic chiasma, its dimensions, the shape of junction of the optic nerves at the chiasma, the diameter, and the length of the intracranial portions of the optic nerves, the length of the optic canals, the antero-posterior width of the pituitary fossa, &c., and states that "while the chiasma does occasionally rest on the optic sulcus, it is nearly always completely posterior to it." "Also, if the chiasma chanced to be situated almost wholly in front of the pituitary body when that organ is diseased, it may possibly explain those cases where the symptom of bitemporal hemianopsia is absent, for tumours of the pituitary body more commonly grow backwards, and erode the dorsum sellæ rather than the anterior part of the fossa." A. NINIAN BRUCE.

PHYSIOLOGY.

SOME EXPERIMENTS ON THE NATURE AND FUNCTION OF
(72) **REISSNER'S FIBRE.** GEORGE E. NICHOLLS, *Journ. of Comp. Neurol.*, 1917, xxvii., Feb., pp. 117-200. (35 figs.)

REISSNER'S fibre is an extremely delicate protoplasmic thread, having, in general, a diameter of more than $1\ \mu$ and less than $3\ \mu$. It possesses a high refractivity, and in the normal tense condition appears to be absolutely structureless. It is normally present in the central nervous system of probably all vertebrates. It extends from the sub-commissural organ in the mid-brain region to the posterior end of the central canal of the spinal cord. Its development is brought about by the confluence of numerous filaments springing from the sub-commissural organ; the composite thread so formed extends backwards into the central canal of the spinal cord. Within the central canal it probably receives numerous additional components from scattered cells in the epithelium which lines the central canal. The fibre is extremely elastic, and appears to exist during life under considerable tension, and is somewhat prone to accidental breakage. In that event, or following artificial section, the free ends may recoil sharply to form tangled knots or "snarls." The retraction is accompanied by a marked increase in the diameter of the fibre.

The writer's experiments were performed on numerous dog-fishes and rays (chiefly *Squalium canicula* and *Raja clavata* and *R. Blanda*). He severed Reissner's fibre at a point quite near to the hinder end of the terminal filament. The result is generally that the fibre is withdrawn in both directions from the lesion, the retraction being apparently effected by a spiral winding of the fibre. In individual rays and dog-fishes in which such retraction of Reissner's fibre has taken place, there is manifested a distinctive reaction: the specimen assumes an abnormal posture while at rest, and probably also exhibits an unusual action when in motion. This reaction occurs very soon after return of the anaesthetised animal to consciousness, and is of variable duration: it is not seen in those individuals in which the fibre has been broken, but has, for any reason, failed to retract. The time required for regeneration is probably not less than a week. Nicholls accepts the suggestion of Dendy (1909) that Reissner's fibre serves to control automatically the flexure and pose of the body. "While it is probable that the related sensory cells are largely concentrated in the sub-commissural organ, it is equally probable that many other such sensory cells are scattered in the ependymal epithelium of the central canal throughout the length of the spinal cord." Nicholls gives a valuable historical summary of the literature, including the very important work of Dendy and Nicholls (1909 to 1913).

LEONARD J. KIDD.

FURTHER OBSERVATIONS ON CEREBRAL HEAT CENTRES.

(73) ERNEST SACHS and P. P. GREEN, *Proc. Amer. Physiol. Soc.*, Dec. 1916; *Amer. Journ. Physiol.*, 1917, xlii, March, p. 603.

"PREVIOUS experiments stimulating the caudate nucleus faradically showed no temperature changes. Since then 93 experiments on cats and rabbits have been carried out. This paper also includes observations on over 150 craniotomies in human beings. There were 31 electrolytic lesions, 16 injection experiments, 40 irrigation experiments after Barbour's method. In the electrolytic lesions temperature rises were noted, but controls showed just as much rise. Injection of emulsion of cortex and caudate into the caudate showed similar temperature changes, but controls showed the same. The results of irrigation by the Barbour method were very variable, and a rise of temperature with cold water, and a fall with hot, was not observed. Irrigation of the liver, using this cannula, gave similar results.

"These experiments do not confirm the view that there is a cerebral heat centre."

LEONARD J. KIDD.

EXPERIMENTAL STUDY OF SECTIONS AND RECOVERIES OF

(74) **NERVES IN DOGS.** (*Contribution expérimentale à l'étude des sections et restaurations nerveuses. Expériences faites sur le chien.*) E. DUROUX and A. COUVREUR, *Presse Médicale*, 1916, Dec. 14, No. 69, p. 572.

CONCLUSIONS :—

(1) From the motor point of view, the dog differs from man; sections of even important nerves, *e.g.*, external popliteal or great sciatic, are followed by merely trifling transient locomotor disturbances.

(2) Immediate restoration of the functions of a divided nerve, after suture, is but illusory. Those only are real which occur after a long interval: in these latter cases, physiology shows, and histological observations confirm the fact, that the peripheral end of the cut nerve does not recover its functions till it has been penetrated by the axons of the central end.

(3) In case of complete section of a nerve, one must perform suturing, and sometimes grafting, if there has been too great a loss of nerve-substance.

(4) One must free a nerve which has been compressed by proliferations of conjunctive tissue, for these hinder the action of the compressed nerve and involve also the functions of other nerves of the limbs.

LEONARD J. KIDD.

EXPERIMENTS ON MOTOR NERVE REGENERATION AND THE
(75) **DIRECT NEUBOTISATION OF PARALYSED MUSCLES BY**
THEIR OWN AND BY FOREIGN NERVES. CHARLES A.
ELSBERG, *Science*, 1917, n.s., xlv., March 30, p. 318.

ELSBERG has performed during the past three years a series of experiments on the nerves and muscles of rabbits' thighs in order to study the question of the physiological regeneration of motor nerves when directly implanted into paralysed muscles, and the possibility of the re-establishment of normal neuro-motor connections. In the first series of experiments all the branches of a nerve to a muscle were cut, and then reimplanted into the same muscle. In the second series, after the wide excision of all the nerves to a muscle, a motor nerve which supplied another muscle was cut and implanted into the paralysed muscles. In all these experiments, after from eight to ten weeks, electrical stimulation of the implanted or reimplanted nerve was followed by a good contraction of the muscle. But when a foreign motor nerve was implanted into a muscle whose normal motor supply was intact, it was found eight to ten weeks later that, while electrical stimulation of the normal motor nerve gave good contraction, similar stimulation of the implanted nerve was without result. Thus hyper-neubotisation of a normal muscle is impossible: a normal muscle cannot be made to take on additional nerve supply. But, if the muscle is permanently separated from its original nerves, then the implanted nerve—which had been hitherto unable to form a connection with the muscle fibres—will establish neuromuscular connections, and electrical stimulation of the nerve will soon cause normal contractions of the muscle. Further experiments showed that, under similar conditions, the normal nerve to a muscle will regain its motor connections with the muscle fibres, and will in some way prevent a foreign nerve which has been implanted at the same time from making any effective neuromuscular connections. Elsberg has been unable to decide whether this is due to a more rapid regeneration of the normal nerve or to the fact that the regenerating normal nerve has an inhibitory influence on the intra-muscular regeneration of the foreign implanted nerve. The experiments are being continued.

LEONARD J. KIDD.

REFLEX CONTROL OF GASTRIC VAGUS-TONUS. F. T. ROGERS,
(76) *Proc. Amer. Physiol. Soc.*, Dec. 1916; *Amer. Journ. Physiol.*, 1917,
xlii., March, p. 605.

"DOGS, in which the splanchnic nerves have been sectioned and with gastric fistulæ, may show hunger contractions after decerebration (Sherrington's method) following a fast of two or three

days. Transection of the brain stem must be done under *deep* anaesthesia. Unless the dog again breathes normally, and unless the pulse rate and strength are nearly normal, the hunger contractions will not appear. Respiratory or cardiac disturbances indicate a quiescent stomach. Pressure effects on the medulla do not seem to stimulate the gastric centres.

"Carlson has called attention to the fact that the hunger contractions are very easily inhibited by various reflexes, but in no case is there a reflex augmentation of these contractions or of gastric tone.

"In a dog, prepared as indicated above, stimulation of the central end of one vagus nerve, with the other nerve intact, is followed by reflex spasmodic contractions of the entire stomach. This effect may be obtained by stimulating the nerve either in the neck or below the diaphragm. In the absence of the splanchnic nerves reflex augmentation of gastric tone and contractions is possible. The vagus contains both the sensory and motor paths of this reflex. This suggests that vagus-tonus, with reference to the stomach, may be a reflex effect similar to that of muscles."

LEONARD J. KIDD.

**ON THE EFFECTS OF OSMOTIC PRESSURE.—IV. THE RELATION
(77) BETWEEN OSMOTIC PRESSURE AND ELECTRIC RESISTANCE OF THE NERVE.** HAJIME TANEMURA, *Acta Scholæ Med. Univ. Imp. in Kioto*, 1916, i., pp. 381-394.

THE change of osmotic pressure changes the volume of the nerve, water and salt ions contained in the nerve trunk, and the degree of its resistance. But the change of this resistance is rather due to the change of content of salt ions than it is due to the change of the volume. The resistance in a unit area of cross-section will be decreased in 1·2 per cent. NaCl to $\frac{1}{3}$ - $\frac{1}{2}$ of what it was in 0·6 per cent. NaCl solution, and it will be increased to 6-12 times or more in distilled water. Such a great change of the resistance will be a great hindrance in determining the irritability of the nerve with inductorium. Therefore, discovery of an accurate method is the first problem to be solved before going into any discussion about the relations of osmotic pressure to the irritability of the nerve.

A. NINIAN BRUCE.

**ON THE SUMMATION OF PROPAGATED DISTURBANCES IN
(78) THE CLAW OF ASTACUS, AND ON THE DOUBLE NEUROMUSCULAR SYSTEM OF THE ADDUCTOR.** KEITH LUCAS, *Journ. Physiol.*, 1917, li., pp. 1-35.

If the nerve to the adductor muscle of the claw in *Astacus* is excited with a single stimulus the muscle may give either a small,

slow contraction or a quick twitch, according to the strength of stimulus used. These are produced by the excitation of different elements in the nerve trunk.

If a first impulse fails to cause a contraction, it yet sends a nervous impulse down the nerve and ensures the success of a second impulse following it at a suitable interval of time. After the relative refractory phase of the first impulse there is a supernormal phase of excitability in which the current required to excite the nerve may be only 80 per cent. of that required to excite the resting nerve. The optimum time for the summation of a second nervous impulse is slightly later than that at which the nerve is at the height of its supernormal phase after the passage of the first impulse.

A. NINIAN BRUCE.

**ON THE LOCALISATION OF THE CALCIUM AND POTASSIUM
(79) SALTS CONCERNED IN THE MEDIATION OF THE ACTION
OF THE VAGUS NERVE ON THE HEART OF THE FROG.**

W. BURRIDGE, *Journ. Physiol.*, 1917, li., pp. 45-50. (2 figs.)

FARADISATION of the base of the frog's ventricle gives effects predominantly inhibitory when the heart contains blood, and effects predominantly excitatory when the heart contains Ringer's solution. After treatment of the perfused frog's heart with potassium salts the capacity to be inhibited from the vago-sympathetic trunk is lost, and the capacity to be inhibited by the faradisation of the local nervous mechanism is regained. The capacity to be inhibited from the vago-sympathetic trunk following treatment with potassium is restored by calcium. It is suggested that the calcium salts, mediating the action of the vagus nerve on the frog's heart, form an integral part of the structure, whatever it may be, on which nicotine acts. The potassium salts concerned in vagus action are situated at the "nerve-endings" in the muscle.

A. NINIAN BRUCE.

**EXPERIMENTAL STUDIES ON THE RELATION OF THE
(80) PITUITARY BODY TO RENAL FUNCTION.** KETIL

MOTZFELDT, *Journ. Exper. Med.*, 1917, xxv., Jan., pp. 153-188.

THE author's conclusions are that the inconstant results of past observations on the relation of pituitary extracts to renal activity have been due chiefly to unsuitable methods. A standard curve of artificially induced polyuria may be plotted for rabbits, giving 200 c.c. of water by mouth. Extracts of the pars intermedia and posterior lobe of the pituitary given by mouth, subcutaneously or intravenously, are able definitely to check polyuria thus induced.

Extracts of the anterior lobe show a similar effect, but only to a slight degree. This antidiuretic effect is constant, and is independent of (a) changes in blood pressure, (b) intestinal absorption, and (c) the vagi. The effect is apparently prevented or delayed by division of the splanchnics, and is diminished by division of the renal nerves near the hilus. A similar antidiuretic property is possessed: (a) by β -imidazolyethylamine, (b) *p*-oxyphenylethylamine, (c) by a preparation from *Secale cornutum*, (d) by small doses of nicotine, (e) by large doses of caffeine, and (f) by extracts of the adrenal cortex. No effect on the polyuria is produced: (a) by strychnine, (b) by morphine, (c) by adrenalin, or by extracts of (d) thyroid, (e) thymus, (f) pineal, (g) pancreas, or (h) corpora lutea. In animals under chloral or paraldehyde anaesthesia a short and inconstant initial increase in flow of urine is seen. The antidiuretic effect is absent or only slightly marked in checking the so-called salt diuresis.

These facts tend to suggest that the antidiuretic action exerted by pituitary extracts on rabbits is caused by stimulation of the sympathetic nervous system, and that the renal vasomotor system in this respect is of chief importance.

Clinically these conceptions bring the polyurias related to disorders of the nervous system and the polyurias of pituitary origin in closer contact.

A. NINIAN BRUCE.

A COMPARATIVE STUDY OF CERTAIN ACTIONS OF
(81) **ADRENALIN IN THE CAT AND RABBIT.** T. S. GITHENS,
Journ. Exp. Med., 1917, xxv., Feb., pp. 323-332. (3 plates.)

THE pupil of the cat is little affected by instillation of adrenalin, but shows a greater responsiveness to adrenalin when given intravenously than does that of the rabbit; the duration of the dilatation effected by intravenous injection is, on the contrary, longer in the rabbit.

In regard to the vasoconstricting effect of adrenalin when administered intravenously, it was found that the intensity as well as the duration of the rise of blood pressure was greater than in the cat.

A. NINIAN BRUCE.

ANTI-NEURITIC SUBSTANCES FROM EGG YOLK. H. STEENBOCK,
(82) *Proc. Amer. Physiol. Soc.*, Dec. 1916; *Amer. Journ. Physiol.*, 1917,
xlii., March, p. 610.

By means of neutral solvents there was prepared a water-acetone soluble fraction from egg yolk, which in small doses, by intraperitoneal injections, was able to cure a pigeon suffering from

polyneuritis. The anti-neuritic principle was found stable to concentrated hydrochloric acid at 98°, and to concentrated alkalis at room temperature, but readily destroyed by dilute alkalis at the boiling temperature. Phosphotungstic acid precipitated it very incompletely. It was not adenine. LEONARD J. KIDD.

PATHOLOGY.

SPHEROIDAL SWELLINGS OF THE AXIS CYLINDERS OF THE
 (83) **CEREBRAL CORTEX.** (Sui rigonflamenti sferoidali dei cilindri della corteccia cerebellare.) ASCANIO ARETINI, *Rassegna di Studi Psichiat.*, 1917, vii., pp. 1-42. (23 figs.)

ACCORDING to the author, the swelling of the axis cylinders of the cerebellar cortex is a primitive manifestation of a pathological change of a retrogressive nature. This may result from any cause, whether physical, toxic, infective, or nutritional, which injures the axones.

The slight and uniform swellings, the elongated, and the single or multiple spheroidal swellings, which the author proposes to call "spheroids," are simply different phases of a reactionary process produced by some pathological stimulus which involves the death of the whole neurone. It may affect a part or the whole of the cerebellar cortex, and has an exclusively pathological significance.

Up till now the author has observed such changes in juvenile and adult general paralysis, cerebral syphilis, Huntington's chorea, arterio-sclerosis, presenile and senile dementia, senility, Alzheimer's disease, cerebellar softening, paralysis agitans, disseminated sclerosis, hydrocephalus, microcephaly, imbecility, epilepsy, encephalomalacia, alcoholism, old cases of dementia præcox, catatonia, cerebral tumour, Little's disease, cerebro-spinal meningitis, acute delirium, and delirium in pneumonia. A. NINIAN BRUCE.

OBSERVATIONS ON BRAIN ATROPHY WITH AND WITHOUT
 (84) **WIDENING OF SULCI.** A. E. TAFT, *Amer. Journ. Insan.*, 1917, lxxiii., Jan., p. 519.

ATROPHY may exist in brains which are not characterised by open sulci; in this paper a report is given of the special examination in six brains, chosen, without respect to clinical or post-mortem findings, from a series of 582. It will be sufficient to give the author's conclusions:—There are probably distinct forms of brain atrophy, one of which is characterised by widening of sulci, and involves the loss of short association fibres which connect adjacent

convolutions. A second, showing no sulcal widening, but some degree of flattening of the gyri, together with notable thinning of the corpus callosum. In addition it may be supposed that with flattening of the gyri and no thinning of the corpus callosum nor widening of the sulci, atrophy may still exist, due to the degeneration of long association fibres. There may be a combination of all or any two of these forms, and some degree of dementia may accompany any of the conditions.

D. K. HENDERSON.

CLINICAL NEUROLOGY.

ASSOCIATED REFLEXES. (De l'association des réflexes.)

(85) AUSTREGESILLO and TEIXEIRA-MENDES, *Revue Neurologique*, Aug. Sept. 1916, pp. 162-169.

THIS article treats on the response by more than one reflex to a single stimulus, illustrative examples being given.

The factors concerned in the production of such phenomena are excessive sensibility of the nerve centres as regards response to stimuli (hyper-reflectivité), and the deviation of diffusion of such response through unusual efferent channels.

Associated reflexes are generally met with in lesions of the medulla and pyramidal tracts, and more rarely in those of the posterior columns of the cord, the nerve roots, and the peripheral nerves.

The associated reflexes represent to reflexes what the synkineses do to movements.

O. P. NAPIER PEARN.

SYNKINESES IN HEMIPLEGIA. (Les syncinésies des hémi-

(86) plégiques.) MARIE and FOIX, *Revue Neurol.*, Aug.-Sept. 1916, pp. 145-162.

THE authors group these associated movements under three headings, and summarise their conclusions on the physiology and pathology of them as follows:—

Total synkinesis (*syncinésie globale*), a raising of the muscular tonus in a hemiplegic limb, is a spasmodic phenomenon almost always associated with contracture, and dependent on a lesion of the pyramidal tracts.

The synkineses of co-ordination are phenomena of medullary automatism. They represent the tendency of the spinal cord to reproduce spontaneously movements habitually associated. These also are connected with a lesion of the pyramidal tracts.

The synkineses of imitation should be divided into two sub-groups, associated movements performed by the healthy side in

accordance with a voluntary movement of the affected side, and *vice versa*. In these a lesion of the pyramidal tracts does not seem to be always essential. They are an exaggeration of the natural tendency towards symmetry in movement.

The second part of the article deals with the practical side of the subject under discussion. It is shown how the presence of synkineses may be either a hindrance or a help to mobility, the latter in cases in which synkineses of co-ordination help towards the automatic execution of habitual movements which have become otherwise impossible. Stress is laid on the diagnostic value of the movements, and a scheme given of the best procedure for eliciting them.

O. P. NAPIER PEARN.

TRACINGS OF PATELLAR CLONUS. (*Ricerche grafiche sul clono (87) della rotula.*) G. ARTOM and C. FRANK, *Riv. di patol. nerv. e ment.*, 1916, xxi., p. 633.

THE writers, as the result of their investigations, came to the following conclusions:—

Patellar clonus is only found in organic lesions; its oscillatory frequency is superior to that of true ankle clonus, and its oscillatory regularity is less perfect. It is easily exhaustible, and presents two forms of exhaustion, one sudden and transitory, the other slow and permanent.

J. D. ROLLESTON.

POSSIBLE PERIODIC VARIATIONS IN THE EXTENT OF THE (88) KNEE JERK IN WOMEN. JESSIE L. KING, *Proc. Amer. Physiol. Soc.*, Dec. 1916; *Amer. Journ. Physiol.*, 1917, xlii., March, p. 607.

A SERIES of daily observations was made on the knee jerks of a group of eleven normal young women, over periods varying from one to five menstrual cycles. The observations covered twenty-nine periods. The writer finds that the evidence points in the direction of the following conclusions:—"That a period of hyper-excitability immediately precedes or accompanies the onset of the menstrual period; that this is followed by a decline in excitability which continues for a few days after the menses have ceased; and that there is then a tendency for it to rise to a slightly higher level than the preceding during the inter-menstrual interval."

LEONARD J. KIDD.

ON THE RÔLE OF DYSTONIA IN THE DISORGANISATION OF (89) VOLUNTARY MOVEMENTS. (*Sur le rôle de la dystonie dans la désorganisation des mouvements volontaires.*) W. VON WOERKOM, *Nouv. Icon. de la Salpêtrière*, 1916-1917, No. 1, p. 37.

THIS paper is not one that can be readily abstracted, and should be read in the original. Some at least of its contentions are open

to criticism, and the anatomical basis assumed for some of the physiological statements is not by any means well founded.

The author takes certain cases of involuntary movement, chorea, athetosis, &c., and tries to show that the defect of volitional movement is due to the abnormal condition of muscle tone in the affected muscle group. This abnormal condition of tone is stated to be of extra-pyramidal type. The author suggests it is of autonomic (sympathetic) origin, and that the corpus striatum, corpus subthalamicum, and mid-brain are possibly centres for sympathetic representation. He suggests that the corpus striatum and regio subthalamica exercise influence on muscular contraction, especially on the tonic element in the same.

S. A. K. WILSON.

HEREDITARY AND FAMILIAL VON RECKLINGHAUSEN'S

(90) DISEASE. J. D. ROLLESTON, *Proc. Roy. Soc. Med.*, 1917, x, (Sect. Study Dis. Child.), p. 32.

THE patients were two girls, aged 19 and 11 years respectively, who had been shown at the Clinical Section of the Royal Society of Medicine six years previously (*v. Review*, 1912, x., p. 1). The eldest had developed since 1911 a large spherical tumour on the inner side of her right upper arm. It was freely movable and quite painless. The molluscous tumours, *café-au-lait* patches, and punctate pigment spots had not shown any decided increase since 1911. In the younger girl the *café-au-lait* patches and punctate pigment spots had increased in number and size, but no molluscum had developed. Rolleston stated that cases of hereditary and familial von Recklinghausen's disease were not very common. In 1900 Alexis Thomson had collected ten such cases, in 1912 another twenty-two had been collected by himself and Dr McNaughtan (*v. Review, ibid.*, p. 8), and since then he had found nine in the literature (all abstracted in the *Review*, 1912-14), a total of forty-one cases.

J. D. ROLLESTON.

CASE OF VON RECKLINGHAUSEN'S DISEASE. J. L. BUNCH, *Proc.*

(91) *Roy. Soc. Med.*, 1917, x, (Sect. of Dermatology), p. 59.

THE patient was a woman, aged 35, with an immense number of molluscous tumours and pigment patches on the body and face, which had appeared ten years previously. There were no tumours on the mucous membranes.

J. D. ROLLESTON.

CASE OF HEREDITARY NEURO-FIBROMATOSIS (VON RECK-

LINGHAUSEN'S DISEASE). E. A. COCKAYNE, *Proc. Roy. Soc. Med.*, 1917, x, (Section Study Dis. Child.), p. 33.

THE mother, aged 35, showed numerous freckles, *café-au-lait* patches on the trunk and limbs, some molluscous tumours on

the left wrist and right hand, and a patch of thickened skin, probably neuro-fibromatous in nature, above the left breast. Of her two children, the elder, a boy aged 5 years, showed *café-au-lait* patches on the trunk, and a molluscous tumour above the left nipple. The younger child, a girl aged 10 months, had several *café-au-lait* patches on the trunk which had been present since birth.

J. D. ROLLESTON.

TABES COMPLICATED BY HEMIPLEGIA FROM CEREBRAL

(93) **SOFTENING.** (*Consideraciones patógenicas y semiológicas sobre un caso de tabes dorsal complicado con hemiplejia por reblandecimiento cerebral.*) E. F. SANZ, *Rev. de med. y cir. práct.*, 1916, cxiii., p. 441.

HEMIPLEGIA is relatively frequent in tabes. It may be early and transient, or late and permanent. A functional and curable form is more frequent. The lesions vary; some are syphilitic, and yield to specific treatment, while others which consist in vascular and cerebral lesions, arterio-sclerosis, softening, &c., resist any such treatment.

Characteristic of tabetic hemiplegia are the rarity of contracture and the absence of the knee and ankle jerks. The re-appearance or non-reappearance of the knee jerk in these cases depends on the relative intensity of the lesions in the posterior columns and the pyramidal tract.

Sanz records a personal case in a lady, aged 55, who, after suffering from tabes for twelve years, suddenly developed right hemiplegia and aphasia. The knee and ankle jerks were absent on both sides, both before and after the ictus.

The case confirms the general rule which obtains in cases of tabetic hemiplegia, especially when the spinal disease is of old standing. The knee jerks remained absent, and the lower limbs, in which hypotonia and inco-ordination had previously been present, showed no contracture, while in the upper limbs, in which these symptoms had been absent, contracture developed.

J. D. ROLLESTON.

A CLINICAL AND ANATOMICAL REPORT OF A CASE OF

(94) **FRIEDREICH'S DISEASE.** W. F. LITCHFIELD, OLIVER LATHAM, and A. W. CAMPBELL, *Med. Journ. of Australia*, 1917, i., Feb. 17, pp. 135-140. (16 figs.)

THE patient was a girl of 8 years. Her parents are healthy. No history of a similar disease in the antecedents or collateral relations. Patient was one of a family of nine—(1) a boy, 14 years, died of this disease; (2) a girl, 12 years, is convalescing from

typhoid fever; (3) a boy, 10 years, is "nervous"; (4) the patient; (5) a girl, 6 years, has twitching of face; (6) a boy, 5 years, has ataxia; (7) a boy, 4 years, healthy; (8) a boy, 3 years, healthy; (9) a baby of 7 months, healthy. The symptoms of the boy of 14 who died from Friedreich's disease began at age of 6 years: he had ataxia of arms and legs, and later, nystagmus: also mental irritability and some speech affection. He died from typhoid fever. The boy of 5 years is living: there was, on admission, a swaying static and dynamic ataxia; slight, but distinct, ataxia of hands; some degree of pes cavus; knee jerks unobtainable; speech normal; no sensory changes nor nystagmus; muscles somewhat slack; shy and uncertain in temper. He showed some improvement on recovery from typhoid, ran about, &c. The patient's symptoms began at age of 6 years; she then walked in a wobbly fashion; later became weaker and thinner, and fell often in the dark. Later could not walk. At time of admission to hospital was irritable, emotional, and weaker; part of the weakness was due to oncoming typhoid fever, from which she died. She had a distinct ataxia, with some jerkiness of movements of hands; speech seemed unaffected. Inconstant nystagmus on lateral deviation; bilateral pes cavus, but no spinal curvature; knee jerk absent, plantars feeble, flexor; some hypotonia of muscles, but no loss of sensibility could be made out (patient only 8 years). Necropsy revealed the usual lesions of typhoid. A description is given of the microscopical appearances of the spinal cord, bulb, and pons, cerebellum, one thoracic dorsal-root ganglion, cortex cerebri, and other parts. Macroscopically, there was a relative diminution in the area of the posterior columns of the cord, and an increase in the ratio of grey matter to white, in contradistinction to what Barker found in his cases. Latham summarises the pathological changes by saying that there is a relatively old sclerosis affecting parts of the posterior tracts, in which the interstitial tissue has assumed an unusual type, resembling that found in disseminated sclerosis. An irregular but less marked sclerosis, of considerable duration, is also present in the lateral regions of the cord. This sclerosis involves areas which do not correspond to sharply defined tracts, and is associated with a much more recent degeneration shown by Marchi staining. There are no marked changes in the pia-arachnoid, blood-vessels, or lymphatics of cord or brain. Only a few cells of Clarke's column, a few of the large cells of the post-central gyrus, and a few cells of the dorsal-root ganglion are affected. The lateral columns are not severely sclerosed; there is no gross atrophy of cerebellum, and the dentate, olivary, and red nuclei are normal. The changes resemble somewhat those of "ataxia paraplegia," especially in the escape of the posterior root zones and ventral fields. Campbell adds some remarks to those

of Latham. As to the paradox of lateral pyramidal sclerosis without paralysis, he says that in all cases examined by himself the sclerosis has not been spread over the whole tract: it has involved only that part which is in contact with the direct cerebellar tract, whereas the inner part has escaped, and to this escape, plus the intact state of the direct pyramidal tracts, is to be attributed the absence of paralysis. As to the origin of the morbid process, Campbell rejects the abiotrophy of Gowers, Newton Pitt's "inherited tendency to general early vascular degeneration," and the German view of a faulty development of the dorsal columns; and he accepts Williamson's view that the parts degenerate because they happen to lie in that segment of the cord wherein the blood supply is weak, viz., in the field supplied by the posterior spinal system of arteries. This important paper should be read *in toto*.
LEONARD J. KIDD.

ON A CASE OF DIPHTHERITIC PARALYSIS. (Sur un cas de (95) paralysie diphtérique.) L. BABONNEIX, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1798.

A PREVIOUSLY healthy man, aged 32, suddenly developed symptoms of acute ascending paralysis with cerebro-spinal lymphocytosis.

The presence of diphtheria bacilli in the throat in spite of an absence of any history of angina, and the improvement obtained by injections of diphtheria antitoxin made Babonneix change his initial diagnosis of Heine-Medin's disease to one of diphtheritic paralysis.

Although it is very rare for diphtheritic paralysis to assume an ascending form in man, this is the usual course in experimental cases, as Babonneix showed in 1903.

Cerebro-spinal lymphocytosis in diphtheritic paralysis has been previously reported by Römheld (*v. Review*, 1908, vi., p. 712), Chauffard and Le Conte (*ibid.*, 1916, xiv., p. 76), and Ravaut and Krolunitsky (*ibid.*, p. 475).
J. D. ROLLESTON.

THE PASSAGE OF NEUTRALIZING SUBSTANCES FROM THE (96) BLOOD INTO THE CEREBRO-SPINAL FLUID IN POLIO-MYELITIS. SIMON FLEXNER and HAROLD L. AMOSS, *Journ. Exp. Med.*, 1917, xxv., April, pp. 499-505.

THE cerebro-spinal fluid taken very early and quite late in the course of acute poliomyelitis exhibits no neutralizing action on filtered poliomyelitic virus. The blood serum on the sixth day of the disease already contains the neutralizing principles. The injection of sterile horse serum into the cerebro-spinal meninges in monkeys increases their permeability, so that they permit the immunity neutralizing principles passively injected into the blood

to pass into the cerebro-spinal fluid. The passage in passively immunized monkeys takes place during a relatively brief space of time, and apparently only while the inflammatory reaction produced by the horse serum is at its height. In monkeys, and probably in man, the intraspinal injection of immune serum in poliomyelitis is curative. In monkeys normal serum exerts no such action, and at present nothing can be stated definitely regarding the therapeutic effect of normal serum in man, except that probably any benefits which may arise from its employment would be attributable, not to the action of the serum as such, but to the escape of circulating immunity principles in the blood made possible by the aseptic inflammation set up by it in the meninges.

As the immunity principles appear in the blood only after several days, and the reported favourable effects of the immune serum treatment relate to the first days of illness, the employment of normal serum is thus not indicated, while that of an immune serum is.

A. NINIAN BRUCE.

**NEUTRALIZATION OF THE VIRUS OF POLIOMYELITIS BY
(97) NASAL WASHINGS.** HAROLD L. AMOSS and EDWARD TAYLOR,
Journ. Exp. Med., 1917, **xxv.**, April, pp. 507-523.

THE results of fifty-six experiments have shown that washings of the nasal and pharyngeal mucosæ possess definite power to inactivate or neutralize the active virus of poliomyelitis. This power is subject to fluctuation in a given person. Apparently inflammatory conditions of the upper air passages tend to remove or diminish the power of neutralization. This neutralizing substance is water-soluble and appears not to be inorganic; it is more or less thermostable, and its action does not depend upon the presence of mucin as such.

It is suggested that the production of healthy carriers through contamination with the virus of poliomyelitis may be determined by the presence or absence of this inactivity or neutralizing property in the secretions. Whether this effect operates to prevent actual invasion of the virus and production of infection can only be conjectured. Probably the property is merely accessory, and not the essential element on which defence against infection rests.

A. NINIAN BRUCE.

**THE RELATION OF THE MENINGES AND CHOROID PLEXUS
(98) TO POLIOMYELITIC INFECTION.** SIMON FLEXNER and
HAROLD L. AMOSS, *Journ. Exp. Med.*, 1917, **xxv.**, April, pp. 525-537.

AMONG the mechanisms which defend the body from infection with the virus of poliomyelitis is the meningeal-choroid plexus complex, which normally is capable of excluding the circulating virus from

the central nervous organs. The complex plays a part also in preventing infection from virus present upon the nasal mucosa. Aseptic fluids which irritate, inflame, or even slightly alter the integrity of the meninges and choroid plexus diminish or remove their protective function. Normal monkey or horse serum, isotonic salt solution, and Ringer's and Locke's solutions, when injected into the meninges, promote infection with the virus introduced into the blood, the nose, or the subcutaneous tissues.

Simple lumbar puncture, and the withdrawal and return of the cerebro-spinal fluid in normal monkeys, hæmorrhage having been avoided, does not promote infection with virus injected into the blood; while the replacement of the cerebro-spinal fluid of one monkey with that of another does, in some instances, lead to infection. Simple lumbar puncture attended with even very slight hæmorrhage opens the way for the passage of the virus from the blood into the central nervous tissues, and thus promotes infection. Hence changes in the structure and function of the meningeal-choroid plexus complex, too slight to be detected by chemical and cellular changes in the cerebro-spinal fluid, or by morphological alterations, suffice to diminish in an essential manner its protective powers.

Of all the irritant fluids tested immune serum alone injected into the meninges is not succeeded by infection from the virus introduced into the blood. The protective property of the immune serum is capable of overcoming the promoting action of normal monkey and horse serum, and the other irritants mentioned.

A. NINIAN BRUCE.

SURVIVAL OF THE POLIOMYELITIC VIRUS FOR SIX YEARS

(99) **IN GLYCEROL.** SIMON FLEXNER and HAROLD L. AMOSS, *Journ. Exp. Med.*, 1917, **xxv.**, April, pp. 539-543. (4 figs.)

SPECIMENS of spinal cord and medulla from human cases and monkeys, which had been kept for four and six years respectively in 50 per cent. glycerol at refrigerator temperature, caused symptoms and lesions identical with those produced by recently collected spinal cord and medulla. The specimens had lost a part of their activity under the conditions described, necessitating larger and repeated doses to induce infection. An ineffective inoculation of tissues containing the virus does not increase resistance, but rather diminishes it, so that a subsequent injection, inadequate in itself, may cause experimental poliomyelitis.

The infectious nervous tissues employed in these experiments did not yield in culture streptococci or other ordinary bacteria.

The power of survival under adverse conditions may not be without significance in respect to the recrudescence of poliomyelitis

in a given locality and after a lapse of years. Hitherto this phenomenon has been accounted for by assuming a fresh importation of a virus of pronounced pathogenic power. Possibly in some cases it is due to the renewed activity of specimens of the virus surviving from a previous epidemic. A. NINIAN BRUCE.

A REPORT ON THE SERUM TREATMENT OF TWENTY-SIX (100) CASES OF EPIDEMIC POLIOMYELITIS. HAROLD L. AMOSS and ALLAN M. CHESNEY, *Journ. Exp. Med.*, 1917, xxv., April, pp. 581-608.

SERUM taken from recently recovered cases of poliomyelitis may be employed in its treatment, and probably yields the best results.

When sterile for ordinary bacteria, free of corpuscles and hæmoglobin, and when injected by the gravity method, observing well-known rules of caution, it may be employed without danger.

The serum should be injected both intraspinally and intravenously, the latter either directly or by way of the subcutaneous tissues.

The earlier in the course of the disease the serum is employed in suitable doses, the more promise there is of benefit. The action of the serum appears to be more precise and definite in arresting paralysis than in rapidly bringing about its retrogression.

The decision to employ the serum should rest upon a clinical examination supported by the results of the microscopic and chemical study of the cerebro-spinal fluid.

The question of multiple and repeated injections of the serum requires further work. Probably in cases in which some degree of muscular weakness develops soon after the injection of serum, reinjection twelve to twenty-four hours later may be advantageous. The milder or less fatal form of poliomyelitis appearing in man is more amenable to the serum treatment than is the highly fatal disease produced by inoculation in monkeys.

A. NINIAN BRUCE.

THE PATHOLOGIC EFFECTS OF STREPTOCOCCI FROM CASES (101) OF POLIOMYELITIS AND OTHER SOURCES. CARROLL G. BULL, *Journ. Exp. Med.*, 1917, xxv., April, p. 557.

WHILE it is not denied that the filterable virus is the inciting cause of poliomyelitis in man and the monkey, its relation to the streptococcus is uncertain.

Streptococci cultivated from the tonsils of thirty-two cases of poliomyelitis were used to inoculate various laboratory animals. In no case was a condition induced resembling poliomyelitis, either clinically or pathologically, in guinea-pigs, dogs, cats, rabbits, or monkeys. On the other hand, a considerable percentage of some

of the rabbits and a smaller percentage of some of the other animals developed lesions due to streptococci. These lesions consisted of meningitis, meningo-encephalitis, cerebral abscess, arthritis, teno-synovitis, myositis, renal abscess, endocarditis, pericarditis, and neuritis. No distinction in the character or frequency of the lesions could be determined between the streptococci derived from poliomyelitic patients and from other sources.

Streptococci isolated from the poliomyelitic brain and spinal cord of monkeys which succumbed to inoculation with the filtered virus failed to induce in monkeys any paralysis or the characteristic histological changes of poliomyelitis. These streptococci are regarded as secondary bacterial invaders of the nervous organs.

Monkeys which have recovered from infection with streptococci derived from cases of poliomyelitis are not protected from infection with the filtered virus, and their blood does not neutralize the filtered virus *in vitro*.

No etiological or pathological relationship between streptococci and epidemic poliomyelitis in man or true experimental poliomyelitis in the monkey could be detected. A. NINIAN BRUCE.

THE CULTIVATION AND IMMUNOLOGICAL REACTIONS OF
(102) **THE GLOBOID BODIES IN POLIOMYELITIS.** HAROLD L.
AMOSS, *Journ. Exp. Med.*, 1917, xxv., April, pp. 545.

THE micro-organism described by Flexner and Noguchi under the name of globoid bodies has been shown to bear a definite relation to epidemic poliomyelitis. The technique employed in the cultivation of these globoid bodies is based upon Noguchi's method of cultivating *Treponema pallidum*.

Two additional cultures of globoid bodies, obtained from the nervous tissues of monkeys in which experimental poliomyelitis was produced, and identical with the original cultures described by Flexner and Noguchi, are reported in this paper. These highly parasitic cultures, like *Treponema pallidum*, are refractory to artificial cultivation. After long cultivation outside the body, the globoid bodies acquire saprophytic properties and grow more readily, provided, however, that they carry a certain quantity of protein matter not denatured.

The rabbit responds slightly with the production of antibodies to the injection of cultures of the globoid bodies; the monkey responds only occasionally, and apparently only when the cultures are injected into the central nervous system. This response is small, and at most leads to slight reactions of agglutination and complement deviation with the cultures. The serum obtained from monkeys recovered from experimental poliomyelitis shows even less agglutination and no complement deviation. The

maximal agglutinative and morphological changes produced in the globoid bodies cultivated in immune monkey serum are obtained in the first generation; after several sub-cultures in the immune serum the reversion to normal takes place in one generation in a non-immune medium. The serum of human beings in the acute or early subacute stages of poliomyelitis gives no complement deviation with the antigen derived from the globoid bodies. The cultivation of the saprophytized globoid bodies through several generations in immune monkey or human serum did not confer upon them pathogenic properties for monkeys. The serum of human beings and monkeys which have survived attacks of poliomyelitis does not fix complement in the presence of antigens prepared from organs of the monkey succumbing to the experimental disease.

The globoid bodies and *Treponema pallidum* present many analogies in cultural, immunological, and pathogenic properties.

A. NINIAN BRUCE.

ON A CASE OF GLIOMA OF THE RIGHT LOBE OF THE CEREBELLUM, WITH SYRINGOMYELIC CAVITIES IN THE SPINAL CORD. (Sopra un caso di glioma del lobo destro del cervelletto con cavità syringomieliche nel midollo spinale.) F. SCHUPFER, *Riv. di patol. nerv. e ment.*, 1916, xxi., p. 644.

THE patient was a girl, aged 16, whose symptoms were headache, vomiting, vertigo, and staggering gait, with tendency to fall to the left. Ophthalmoscopy showed the presence of choked disc most marked in the right eye. Muscular atrophy was pronounced, especially in the upper limbs, where it assumed the Aran-Duchenne type, and was associated with scoliosis with a convexity to the right, and a diminution of sensibility to pain in the four limbs, in which, however, not only tactile but also thermal sensibility was normal.

J. D. ROLLESTON.

MELANOTIC TUMOUR OF THE BASE OF THE BRAIN INVOLVING THE RIGHT FACIAL, ACCOMPANIED BY FLACCID PARAPLEGIA AND MENINGEAL IRRITATION IN ITS VICINITY. SARCOMATOSIS OF THE PIA MATER AND NEURAL PARAPLEGIA. (Un cas de tumeur mélanique de la base du cerveau intéressant le facial droit s'accompagnant de paraplégie flasque et d'irritation méningée de voisinage. Sarcomatose de la pie-mère et paraplégie neurale.) E. LENOBLE and R. INIZAN, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1511.

THE patient was a man, aged 68. The lesion did not cause any severe disturbance until about three weeks before death, when violent headache and vomiting suggested a cerebral tumour. A week before death paraplegia suddenly developed.

The autopsy showed generalization of a sarcoma of the orbit to the pia mater throughout the whole of its extent, including its prolongations along the lumbo-sacral nerve roots. Sections of the nerve centres at the different levels showed that they were not responsible for the paraplegia, whereas examination of the nerve roots revealed very extensive disease. J. D. ROLLESTON.

FAMILIAL CIRRHOSIS OF THE LIVER: FOUR CASES OF
(105) **ACUTE FATAL CIRRHOSIS OF THE LIVER IN THE**
SAME FAMILY, &c.: SUGGESTED RELATIONSHIP
TO WILSON'S PROGRESSIVE DEGENERATION OF THE
LENTICULAR NUCLEUS. BYROM BRAMWELL, *Edin. Med.*
Journ., 1916, Aug.

DR BRAMWELL describes four cases of acute fatal cirrhosis of the liver occurring in a brother and three sisters, aged 9, 10, 14, and 14 years respectively. In each case the disease was acute, and rapidly fatal in some three or four weeks. The liver symptoms filled the clinical picture, and no nervous symptoms were noted. There was no evidence of syphilis or of alcohol.

Dr Bramwell suggests that his familial cases without nervous symptoms are perhaps merely a preliminary and not fully developed stage of the disease which Wilson has termed progressive lenticular degeneration. Wilson's most acute cases had terms of four, six, and thirteen months respectively, while the cases here described were so acute that Dr Bramwell suggests the nervous symptoms had not time to develop. S. A. K. WILSON.

THE PRODUCTION IN DOGS OF A PATHOLOGICAL CONDITION
(106) **WHICH CLOSELY RESEMBLES HUMAN PELLAGRA.**
RUSSELL H. CHITTENDEN and FRANK P. UNDERHILL, *Proc.*
National Acad. Sciences of U.S.A., 1917, iii., March, p. 195.

THE writers have produced in dogs a diseased state closely resembling human pellagra, by feeding them on a diet of boiled (dry) peas, cracker meal, and cotton-seed oil, or lard. Ingestion of suitable quantities of meat causes the symptoms of disturbed nutrition to disappear. On the other hand, if the amount of meat contained in a selected mixed diet is insufficient, the same evidences of abnormality may appear. The onset is generally sudden—the dog refuses to eat, but appears normal on examination; he lies quiet, but apathetic. After a day or two of refusal of food, his mouth becomes covered with foul pustules: its mucous lining comes off in shreds if lightly stroked: intense salivation: teeth normal: foul, bloody, frequent diarrhoea. There may be

pustules on thorax and upper abdomen. Death follows without other striking features. Occasionally, however, convulsions occur, either with or without the symptoms just detailed. Necropsy reveals one of two types of lesions:—(1) In animals showing foul mouth and bloody diarrhoea there is an intense hæmorrhagic appearance of the lower bowel and rectum; (2) in those which die rapidly from convulsions, the only abnormality of the alimentary tract consists of one or more large ulcers of the duodenum. The writers add that the typical symptoms may be induced in dogs, but with much greater difficulty, when a diet containing meat, cracker meal, and lard is fed in appropriate quantities. For the production of the diseased condition the meat intake must be reduced to a certain undefined minimum. They conclude that this abnormal state may be referred to a deficiency of some essential dietary constituent or constituents, presumably belonging to the group of hitherto unrecognised but essential components of an adequate diet.

LEONARD J. KIDD.

GENITO-GLANDULAR DYSTROPHY. (*Dystrophie génito-glandulaire*) DE SOUZA and DE CASTRO, *Nouv. Icon. de la Salpêtrière*, 1916-17, No. 1, p. 1.

THIS is a long, well documented, and well illustrated article on different varieties of genito-glandular dystrophy. As it constitutes only the first part of a longer communication promised by the authors, a more detailed review may be for the time postponed.

S. A. K. WILSON.

LIPODYSTROPHIA PROGRESSIVA. F. PARKES WEBER, *Quart. Journ. Med.*, 1916-17, x., p. 131.

THIS term, which was first introduced by G. A. Simons in 1911, is applied to a rare disease possibly confined to the female sex, and characterised by the progressive disappearance of the subcutaneous fat from the parts above the lower extremities.

The fat atrophy first appears in the face, and then spreads to the trunk and upper extremities. The lower extremities and buttocks are never involved. The general health is not affected.

The ætiology is unknown, but an endocrinic origin has been suspected. The disease generally commences in childhood, especially at the age of six years.

No remedy has been found for the fat atrophy; only temporary improvement results from paraffin injection. The writer refers to two cases of his own, and to other recorded cases.

J. D. ROLLESTON.

THE DEATH OF VOLTAIRE. (*La mort de Voltaire.*) R. BOISSIER, (109) *Thèses de Paris*, 1916-17, No. 8.

THE clinical history and post-mortem findings indicate that Voltaire's death was due to uræmia following on cystitis, secondary to hypertrophy of the prostate. The legend of his terminal coprophagia was an invention of his enemies.

J. D. ROLLESTON.

CYTOLOGY OF THE CEREBRO-SPINAL FLUID IN THE COURSE
(110) **OF A MALARIAL ATTACK.** (*Cytologie du liquide céphalo-rachidien au cours de l'accès palustre.*) MONIER - VINARD, PAISSEAU, and H. LEMAIRE, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1607.

THE writers found that in twenty-three out of thirty-three specimens of the cerebro-spinal fluids removed during a malarial attack, there was a meningeal cellular reaction. The polymorphs were very rarely affected, the reaction consisting chiefly in lymphocytosis and mononucleosis associated with endothelial cells. The albumin of the cerebro-spinal fluid was simultaneously very much increased. Apart from the meningeal states in which it was constantly present, the reaction was sometimes found in other conditions, especially during the presence of herpes, amaurosis, or paralysis of a cranial nerve.

J. D. ROLLESTON.

MENINGEAL REACTIONS IN ICTERO-HÆMORRHAGIC SPIRO-
(111) **CHÆTOSIS. VIRULENCE OF THE CEREBRO-SPINAL FLUID.** (*Réactions méningées dans la spirochétose ictéro-hémorragique. Virulence du liquide céphalo-rachidien.*) S. COSTA and TROISIER, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1802.

A MAN, aged 41, suffering from febrile icterus developed headache, nuchal rigidity, and Kernig's sign. Lumbar puncture gave issue to a clear fluid under tension, containing albumin and with a marked cellular reaction (mononuclears and lymphocytes). Intra-peritoneal injection of the spinal fluid into a guinea-pig produced a fatal icterus, in which the *S. ictero-hæmorrhagica* was found in the organs post mortem.

J. D. ROLLESTON.

THE SIGNIFICANCE OF CHANGES IN CELLULAR CONTENT
(112) **OF CEREBRO-SPINAL FLUID IN NEUROSYPHILIS.** HARRY C. SOLOMON and HILMAR O. KOEFOD, *Boston Med. and Surg. Journ.*, 1915, clxxiii., Dec. 23, pp. 996-1001.

THE present study consists in interval cell counts in forty-six cases diagnosed as general paresis or cerebro-spinal syphilis.

Treatment, consisting of mercury, intravenous salvarsan, and potassium iodide, was given in nineteen cases; the other twenty-seven received no treatment.

The conclusions are as follows:—

The number of cells found in the fluid of untreated cases gives no definite information of prognostic value. It does not help in differentiating between cerebro-spinal syphilis and general paralysis, and does not indicate if the process has been active or severe. Cases showing natural remissions may show no reduction in the cell count or other spinal fluid findings. Cases treated with salvarsan, either intraspinally or intravenously, tend to show a more or less rapid fall in the cell count. This count as a rule remains low during treatment, but is likely to rise when treatment has been discontinued, and may rise during treatment after having just fallen. Cases may show remissions during treatment, and still have a pleocytosis. Treated cases having the cell count fall to normal may at the same time become very much worse, and develop more marked paralytic symptoms. In general paralysis the cell count in no way parallels the other spinal fluid findings. The change in cell count seen in syphilitic disease untreated is also found in non-syphilitic diseases, as brain tumour. It offers nothing of prognostic importance in syphilis of the nervous system unless accompanied by improvement of the other laboratory signs. It is not an index to the predominance of irritative or degenerative changes.

A. NINIAN BRUCE.

DIAGNOSTIC VALUE OF LANGE'S GOLD SOL TEST (based on (113) 500 examinations of the spinal fluid). HARRY C. SOLOMON, HILMAR O. KOEFOD, and EDWARD S. WELLES, *Boston Med. and Surg. Journ.*, 1915, clxxiii., Dec. 23, pp. 956-960.

FLUIDS from cases of general paresis will give a strong and fairly characteristic reaction in most cases, especially if more than one sample is tested. A weaker reaction is very rare.

Fluids from cerebro-spinal syphilis often give a weaker reaction than the paretic, but in a fairly high percentage of cases give the same reaction as the paretics.

Non-syphilitic cases may give the same reaction as the paretics; these cases are usually chronic inflammatory conditions of the central nervous system.

When a syphilitic fluid does not give the strong "paretic reaction" it is good presumptive evidence that the case is not general paresis, and this test offers a very valuable differential diagnostic aid between general paresis, tabes, and cerebro-spinal syphilis.

Light reactions may occur without any evident significance,

while a reaction of no greater strength may mean marked inflammatory reaction.

Tuberculous meningitis, brain tumour, and purulent meningitic fluids characteristically, but not invariably, give reactions in higher dilutions than syphilitic fluids.

The unsupplemented gold sol test is insufficient evidence on which to make any diagnosis, but used in conjunction with the Wassermann reaction, chemical and cytological examinations, it offers much information, aiding the differential diagnosis of general paresis, cerebro-spinal syphilis, tabes dorsalis, brain tumour, tuberculous meningitis, and purulent meningitis.

The authors believe no cerebro-spinal fluid examination is complete for clinical purposes without the gold sol test.

A. NINIAN BRUCE.

THE DEVELOPMENT OF THE GOLD SOL "PARETIC" REACTION AS COMPARED WITH THE "CEREBRO-SPINAL SYPHILITIC" TYPE, CONSIDERED FROM THE TIME NECESSARY TO FORM A COMPLETED REACTION. H. C. SOLOMON and E. S. WELLES, *Boston Med. and Surg. Journ.*, 1916, clxxiv., Jan. 13, p. 50.

THE "cerebro-spinal syphilitic" type of colloidal gold reaction is strongest in the third, fourth, and fifth tubes grading off in the first and second tubes, and those of the high dilution. Less characteristic is a curve reaching its height in the first tube, but not going as high as 5 + reaction.

In many "paretic" reactions it will be noticed that the reaction begins most strongly in the third, fourth, and fifth tubes, and is weaker in the first and second, and in the higher dilutions, so that it, as it were, passes through a stage identical with the cerebro-spinal type of reaction before developing the complete paretic result. The cerebro-spinal type may thus be regarded as a *forme fruste* of the paretic reaction.

A. NINIAN BRUCE.

VARIETIES OF THE GOLD SOL TEST (LANGE) IN SEVERAL LOCI OF THE CEREBRO-SPINAL FLUID SYSTEM: A STUDY OF TWENTY-EIGHT AUTOPSIED CASES. H. C. SOLOMON, *Boston Med. and Surg. Journ.*, 1917, clxxii., April 29, pp. 625-629.

FLUID was obtained from the following cisternæ and spaces in a series of 28 cases post mortem (*v. Review*, 1915, xiii., p. 603), viz.:—(1) Cerebral subarachnoid space, (2) epicerebral space, (3) lateral ventricles, (4) third ventricle, (5) cisterna at base of brain, and (6) spinal subarachnoid space, and tested by the colloidal gold method.

It was found that in 24 of these 28 cases the reaction differed

in fluids from the same case obtained from different loci, while in 4 the results were identical. In 2 the reaction was identical in two loci, while differing in other loci.

In one case of general paralysis the spinal fluid gave the reaction 5555555543, while the fluid from the third ventricle showed 0122332210. Another case of general paralysis gave the following reactions: spinal fluid 5555544432, fluid from base of brain 3334452211, fluid from subdural space 3332221000, and fluid from the third ventricle 0001100000. A hydrocephalic imbecile with cerebro-spinal tuberculous meningitis gave, with the fluid from the lateral ventricle, 002221000; fluid from the base of the brain, 0000012210; and with the spinal fluid, 111111223.

It would thus appear that the examination of the fluid obtained by lumbar puncture is not in all cases a true indication of the condition existing higher up, and that in all probability there is not a free circulation or exchange of the fluid.

A. NINIAN BRUCE.

LATENT NEUROSYPHILIS AND THE QUESTION OF GENERAL (116) PARALYSIS—SINE PARESIS. E. E. SOUTHARD and H. E. SOLOMON, *Boston Med. and Surg. Journ.*, 1916, clxxiv., Jan. 6, pp. 8-15.

THERE is a group of cases showing the laboratory signs characteristic of central nervous system syphilis, viz.:—(a) Positive Wassermann reaction in the serum; (b) positive Wassermann reaction in the spinal fluid; (c) pleocytosis; (d) excess of globulin and (e) of albumen in the spinal fluid; (f) gold sol reaction of central nervous system syphilis, and which show no sign or symptom of neural syphilis. The authors believe such cases represent a form of chronic cerebro-spinal syphilis, probably paretic in type. They have the greatest theoretical and practical significance in the conception of the life history of neural syphilis, in the concept of allergie, in regard to results of treatment, and finally as to the evaluation of the laboratory tests. They may also be the final links in the chain connecting the symptoms of primary syphilis with cerebro-spinal syphilis and general paresis.

A. NINIAN BRUCE.

THE FACTORS WHICH GOVERN THE PENETRATION OF (117) ARSENIC (SALVARSAN) AND ANILINE DYES INTO THE BRAIN, AND THEIR BEARING UPON THE TREATMENT OF CEREBRAL SYPHILIS. JAMES M'INTOSH and PAUL FILDES, *Brain*, 1916, xxxix., pp. 478-483.

CERTAIN dye substances can pass directly from the blood to the brain substance proper without being found in the cerebro-spinal fluid, while others fail to penetrate into the brain.

The chief factor which governs the passage of the dyes is their solubility reactions. This is a peculiar solubility, and not a general lipid solubility. It corresponds to a solubility in chloroform and in water, or perhaps to their partition coefficient in these liquids.

The present-day arsenical remedies are, to some extent, inefficient in the treatment of syphilis of the central nervous system, because they do not possess the necessary solubility to allow them to pass from the blood-vessels into the brain substance. Their relative inefficiency has nothing to do with their absence from the cerebro-spinal fluid.

The dyes here used were methylene blue, neutral red, fluoresceine, and indigo-carmin. These were injected intravenously into rabbits, the animal being killed about five minutes later by air embolism.

A. NINIAN BRUCE.

THE TREATMENT OF SYPHILIS OF THE CENTRAL NERVOUS
(118) **SYSTEM WITH INTRASPINAL INJECTIONS OF MERCURIALIZED SERUM.** JULIAN MAST WOLFSOHN, *Amer. Journ. Med. Sci.*, 1917, cliii., Feb., p. 265.

THE facts of greatest interest to the neurologist in connection with the intraspinal mercurialized serum treatment are:—

1. There is no danger in its administration.
2. For local treatment it is very efficacious in syphilis of the central nervous system, especially in the treatment of tabes dorsalis, in which lancinating pains are the predominant symptom.
3. Due to its stability, the serum may be used at any time after its preparation.
4. The lack of expensive drugs used in its preparation makes it invaluable at present.
5. There is no objection to a combined salvarsanized and mercurialized treatment.
6. It must not be concluded, from the short space of time (eight months) that has elapsed since the beginning of this form of treatment in these cases, that relief is going to be permanent. From the results obtained so far it certainly has mitigated the symptom of pain.

A. NINIAN BRUCE.

MERCURIALIZED SERUMS. F. E. STEWART, *N.Y. Med. Journ.*, 1917, (119) cv., p. 121.

CORROSIVE sublimate becomes non-corrosive and non-irritating when dissolved in normal serum. The compounds thus formed

are just as toxic, and probably therapeutically as efficacious, as mercuric bichloride itself. When prepared from heterologous serums, mercurialized serums must be regarded as heterologous serum preparations, requiring conformity to the same rules in their administration as applied to other heterologous serums, such as diphtheria antitoxin and antibacterial serums. Mercury in the form of mercurialized serums is an ideal form for administering mercury subcutaneously, intramuscularly, intravenously, and intraspinaly. Subcutaneous or intramuscular administration is the method of choice. Intravenous or intraspinal administration should only be resorted to when specially indicated.

A. NINIAN BRUCE.

**ACUTE INFLAMMATION OF THE SUPRARENALS IN SECOND-
(120) ARY SYPHILIS FOLLOWING ERYTHEMA MULTIFORME.**

(Surrénalite aiguë dans le cours d'une syphilis secondaire et à la suite d'un erythème polymorphe.) P. BLUM, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1767.

THE patient was a woman, aged 25, in whom the symptoms of vomiting, asthenia, hypotension, hypothermia, and the presence of Sergent's white line (*v. Review*, 1907, v., p. 324) suggested acute inflammation of the suprarenals. Recovery followed oral administration of adrenalin.

J. D. ROLLESTON.

THE SYNDROME OF THE POSTERIOR LACERATE FORAMEN.

(121) (Sur le syndrome du trou déchiré postérieur.) M. VERNET, *Paris Méd.*, 1917, vii., p. 78.

A MAN, aged 52, shortly after the appearance of a labial chancre, developed enlargement of the glands on the corresponding side of the neck, and at the same time and on the same side paralysis of the internal branch of XI. (hemiparalysis of the soft palate and larynx, with acceleration of the pulse), paralysis of IX. (disturbances of taste in the posterior part of the tongue, and paralysis of the superior constrictor of the pharynx), and paralysis of X. (sensory disturbance of palate, pharynx, and larynx, and disordered salivation), although the neighbouring cranial nerves, especially V., VII., VIII., and XII., were unaffected. Specific treatment produced a simultaneous improvement in the glandular enlargement and the nerve symptoms. Vernet regards the compression caused by the glandular enlargement in the region of the posterior lacerate foramen as the cause of the nervous symptoms.

J. D. ROLLESTON.

THE SYPHILIS PROBLEM AMONG CONFINED CRIMINALS.

(122) E. N. BOUDREAU, *Med. Record*, 1916, xc., p. 981.

BOUDREAU summarizes his paper as follows:—

1. 16·85 per cent. of the males, and 33·85 per cent. of the females of Auburn Prison are found to have given a positive Wassermann.

2. 7·5 per cent. of all admissions are potential sufferers from paresis or tabes, or some other form of nervous syphilis, and consequently future wards of the State.

3. It would cost the State of New York approximately \$9,000 a year to treat properly all the cases of syphilis at Auburn Prison.

4. History, glandular enlargement, and physical findings in general are further proven to be lacking as evidence of the presence of syphilis.

5. Epitrochlear gland enlargement is not pathognomonic of syphilis.

J. D. ROLLESTON.

A STUDY OF VON DUNGERN'S INDIGO TEST FOR SYPHILIS.

(123) EDWARD P. FLOOD, *Journ. of Immunol.*, 1916, ii., Dec., pp. 69-74.

VON DUNGERN in 1915 described an alkaline indigo solution that, when added to an inactivated serum, would inhibit its normal coagulation by heat. He believed that the indigo present was the active inhibiting substance. It is here shown that neither the indigo nor the salt content of the reagent takes part in the inhibition of the coagulation. The alkali is the sole inhibiting agent.

Von Dungern further states that with similar amounts of the reagent this inhibition was less marked in syphilitic than in normal sera. It was here found that such a difference was present in but very slight degree and with no constancy.

The method is of no value in the serum diagnosis of syphilis, for in a series of 140 sera, 40 of which were evidently syphilitic, coagulation occurred in but 3 with the prescribed amount of reagent.

A. NINIAN BRUCE.

**THE HECHT (GRADWOHL MODIFICATION) COMPLEMENT
(124) FIXATION REACTION IN SYPHILIS, WITH SPECIAL
REFERENCE TO CHOLESTERINIZED ANTIGENS.**

JOHN A. KOLMER, *Journ. of Immunol.*, 1916, ii., Dec., pp. 23-37.

THE Hecht reaction as modified by Gradwohl, and conducted with a properly standardized antigen, constitutes a very delicate control over the Wassermann reaction.

It possesses a greater negative than a positive value, because it

is open to the error of falsely positive or proteotropic reactions, as obtained in the Noguchi test employing active or unheated serum. The Hecht reaction yielded about 4 per cent. of falsely positive or pseudo-reactions with one or more of the three different antigens used in this study; the fewest of these pseudo-reactions occurred with an extract of acetone-insoluble lipoids. The antigen or antigens employed in the modified Hecht reaction must be carefully standardized at frequent intervals, in order to reduce the percentage of pseudo-reactions to a minimum. A suitable extract of acetone-insoluble lipoid of heart muscle generally proved the superior antigen for the Hecht-Gradwohl reaction. An alcoholic extract of syphilitic liver proved of least antigenic sensitiveness, and yielded the highest percentage of false or pseudo-reactions. The modified Hecht and Wassermann reactions, including the use of cholesterinized extracts, yielded similar results in 82 per cent. of a series of 360 sera.

The modified Hecht reaction yielded about 16 per cent. more positive reactions, and about 12 per cent. more *true* positive reactions, than the Wassermann reaction conducted with a cholesterinized extract as antigen; 26 per cent. more *true* positive reactions than with an alcoholic extract of liver; and 18 per cent. more *true* reactions than with an extract of acetone-insoluble lipoids.

The modified Hecht reaction has its greatest value as a serological control in the treatment of syphilis. During treatment the Wassermann reaction with an alcoholic extract of syphilitic liver is usually extinguished first; after considerable more treatment it is generally extinguished with a cholesterinized extract; the Hecht reaction is usually last to react in a negative manner.

The author thinks that suitable and standardized cholesterinized alcoholic extracts of heart muscle constitute the most sensitive antigens in the Wassermann reaction. It is a serious error to regard a person as cured of syphilis on the basis of a negative Wassermann reaction conducted with an alcoholic extract of syphilitic liver alone.

A. NINIAN BRUCE.

A COMPARATIVE STUDY OF DIFFERENT METHODS OF (125) PERFORMING THE WASSERMANN TEST FOR SYPHILIS.

J. WHEELER SMITH and WARD J. MACNEAL, *Journ. of Immunol.*, 1916, ii., Dec., pp. 75-93.

IN a group of 110 syphilitic individuals in various stages of the disease, the cholesterin-reinforced antigen with incubation at 37° C. gave 58.2 per cent.; the simple antigen at 37° C., 32.2 per cent.; and the simple antigen at 8° C., 77.1 per cent. of positive tests.

In a group of 43 patients, probably syphilitic, the first method

yielded 65·9 per cent., the second 36·3 per cent., and the third method 75·0 per cent. of positive fixations.

In a group of 59 patients, probably not syphilitic, the first method yielded 40·0 per cent., the second 1·6 per cent., and the third 5·0 per cent. of positive tests.

In a group of 265 non-syphilitic patients, tests by all methods were negative throughout.

The use of a simple alcoholic antigen, with the first incubation carried out in the ice-box for four to twenty-four hours, is more sensitive in the detection of syphilis than the other procedures tested. Furthermore, a positive result thus obtained is much more trustworthy evidence of syphilis than is a positive fixation with a cholesterinized antigen.

A. NINIAN BRUCE.

A STUDY OF TWO HUNDRED AND NINETY POST-MORTEM

(126) **WASSERMANN REACTIONS.** STUART GRAVES, *Journ. of Immunol.*, 1916, ii., Dec., pp. 75-93.

POST-MORTEM Wassermann reactions confirmed ante-mortem reactions in 95 per cent. of 38 control cases. Positives were confirmed in serum six hours post mortem and negatives in serum twenty-two hours post mortem.

In 90·4 per cent. of cases showing post-mortem lesions of syphilis, or presenting positive evidence of syphilis in their histories, the sera post mortem gave positive Wassermann reactions. Only 7 per cent. of 282 cases showed negative reactions in the presence of anatomical lesions (aneurysms) characteristic of syphilis.

The average percentage of specific reactions in this series was almost as high post mortem as would be expected ante mortem. The Wassermann reaction thus performed on post-mortem blood is a reliable aid to the diagnosis of syphilis.

A. NINIAN BRUCE.

CORRECT AND INCORRECT METHODS OF PERFORMING THE (127) **"DAILY TITRATIONS" FOR THE WASSERMANN RE-** **ACTION AND OTHER FORMS OF COMPLEMENT FIXATION.**

REUBEN OTTENBERG, *Journ. of Immunol.*, 1916, ii., Dec., pp. 39-46.

EXPERIMENTS are recorded showing that in the Wassermann reaction the method used by many workers of compensating for increased hæmolytic strength of the complement, by using a diminished amboceptor dose, inevitably leads to a negative result in certain grades of weakly positive sera. The correct method of compensating for complements of greater than average hæmolytic

strength is by using a smaller dose of complement. The method of doing daily complement titrations followed by many workers is greatly to be preferred, therefore, to the method of keeping the dose of complement fixed, and doing daily amboceptor titrations; and undoubtedly the differences between these two methods are partly responsible for the different results that have been reported from different laboratories on identical sera.

A. NINIAN BRUCE.

EPILEPSY: A METABOLIC DISEASE. GUY P. U. PRIOR and (128) S. EVAN JONES, *Journ. Ment. Sci.*, 1917, lxiii., Jan., p. 36.

730 records of the calcium excretion in the urine of 19 patients showed a low excretion, but a rise occurred some days before a series.

There was no constant change in the calcium blood index in 18 patients before a fit, but the index was always high after a serial attack.

The excretion of phosphorus in the urine of women was uniform, whereas it was irregular in the men, and was diminished before a series of fits.

464 examinations on the coagulation time of the blood of 76 patients gave a shortening before a fit, a greater shortening before a series, and in both cases a lengthening afterwards.

290 observations on the alkalinity of the blood of 50 patients elicited, as a rule, a low alkalinity, especially before a fit.

A leucocyte count (395 examinations) was generally low before a fit and rose later, the rise being more marked after a series, and the rise was almost entirely due to an increase in polymorphonuclear leucocytes.

The systolic blood pressure of 64 men and 51 women was equal or higher in the recumbent position than when standing. The authors are unable to explain this.

Of 30 patients treated with calcium, either alone or with one or more of the ductless glands, 5 exhibited no change, 13 have had their fits halved, 12 have shown a lesser reduction, their fits were less severe, and they were mentally brighter. The women showed more improvement, and their catamenia were more regular.

Of the ductless glands, the thymus appeared to be of most use.

Serial attacks can be cut short by subcutaneous injections of 10 gr. of calcium iodide (made up in solution of glucose in salt solution, 2 gr. to the c.c.), and by intravenous injections of calcium chloride. (Calcium lowers the excitability of nerve and muscle, whereas sodium salts enhance excitability, and if this be so the

beneficial results of salt-free diet and calcium treatment in epilepsy are explicable by a substitution of calcium for sodium.)

The periodic crises of epilepsy represent an exaggerated psychomotor reaction, there being an afferent impulse which excites a wide area of the cortex, and causes unconsciousness and an uninhibited motor excitation, the abeyance of inhibition being the consequence of deficient resistance or refractivity at the synapses, and due probably to disordered calcium metabolism.

The organs responsible for the disturbance of calcium metabolism may be found in the ductless glands. Changes are found in these glands, the most frequent being persistence and enlargement of the thymus. (*Cf. Review*, 1917, xv., p. 34.)

H. DE M. ALEXANDER.

**PRELIMINARY REPORT OF FOUR CASES OF STATUS
(129) EPILEPTICUS TREATED WITH SUCCESS. WILLIAM HELD,**
Chicago Med. Recorder, 1917, xxxix., March, p. 110.

ARGUING that if a certain form of treatment was beneficial in a few attacks it ought to be, or might be, good in a series of continuous attacks, Held treated six cases of status epilepticus thus:—Hot applications to feet, cold to head; patient's body covered, and windows opened; as far as possible, phlegm was removed from mouth. A high warm water enema. With assistance of one person to hold the arm, Held removed 10 to 20 c.c. of patient's blood; the larger quantity was taken when head congestion was great. The blood was at once centrifuged, after having been chilled under a stream of running cold water, or ice when obtainable. The serum was drawn off and prepared as follows:—To 2 c.c. of the serum were added 2 c.c. of sterile distilled water (physiological salt solution). This was shaken a few times, and one-half the quantity poured out and thrown away. To the remaining 2 c.c. of solution, again 2 c.c. of salt solution was added, shaken, and half of it discarded. This process was repeated until 15 times 2 c.c. of the salt solution had been added. To the 4 c.c. of solution finally obtained Held added one drop of dissimilar anti-epileptic ferment from any epileptic under treatment. Of this solution, *i.e.*, the patient's blood serum, the physiological salt solution, and the anti-epileptic ferment, he injected 5 drops intravenously. His results were as follows:—In one case the anti-epileptic serum was not added. This and one other case died without noticeable change in their condition following injection or treatment. Four cases survived the "status" after the treatment. In two of these patients the regular appearance of their epileptic attacks was delayed by five weeks following their recovery from "status." Following the injection, the patients did not have any convulsive seizure after the state of quietus immediately following

the injection. Held advises that, as the injections are intravenous, the procedure should be carried out under aseptic conditions, and preferably by one who is familiar with specific serum therapy.

LEONARD J. KIDD.

**THE RELATION BETWEEN THE GENETIC FACTORS AND THE
(130) AGE OF ONSET IN ONE HUNDRED AND FIFTY-SEVEN
CASES OF HEREDITARY EPILEPSY.** D. A. THOM, *Boston
Med. and Surg. Journ.*, 1915, clxxiii., Sept. 23, pp. 469-473.

As the result of a study of 157 cases (75 females and 82 males) from the Monson State Hospital, including all the hereditary cases of epilepsy where complete family histories were available, the following conclusions were drawn:—

In the order of potency in the production of an early onset of epilepsy in the offspring, feeble-mindedness came first, followed in order by migraine, epilepsy, alcohol, and insanity. With the exception of alcoholism, all the maternal defective factors were manifested in the offspring at an earlier date in the form of epilepsy than those factors transmitted by the father. When both parents had the same defect the onset was at an earlier date than in those cases where only one parent was defective. There was practically no difference between the average age at onset in those cases inheriting two or three defects and in those cases where the family history revealed but one. Those cases with direct heredity (parents or grandparents) averaged an earlier onset than those cases with collateral heredity. The average age of onset was a little over eight years younger than 205 cases computed without reference to heredity. 39 per cent. of the entire group had more than one hereditary defect in the family history. 80 per cent. had direct heredity, and 58 per cent. of the entire group gave a history of epilepsy in some member of the family. 11 cases, the offspring of epileptic parents and the parents of an epileptic offspring, were free from mental defects themselves.

A. NINIAN BRUCE.

CHOREA RHYTHMICA IN A MAN. F. PARKES WEBER, *Proc. Roy. Soc. Med.*, 1916-17, x. (Clin. Sect.), p. 6.

THE patient, a Polish Jew, aged 38, showed rhythmic movements of the neck and both arms, which increased in rate and amplitude and force under observation, but quieted down when he was left alone, and ceased entirely during sleep.

Psychical factors connected with the war played an important part in the case.

J. D. ROLLESTON.

ACUTE POLYNEURITIS (OF THE ALCOHOLIC TYPE, BUT NOT (132) DUE TO ALCOHOLISM), RAPIDLY RECOVERED FROM, IN A PATIENT WITH CHRONIC DIABETES MELLITUS WITHOUT HYPERGLYCAEMIA. F. PARKES WEBER, *St Bart.'s Hosp. Rep.*, 1916, I., Part II., p. 163.

THE patient was a woman, aged 40, who had probably had glycosuria continuously since she was 13 years old. The attack of acute multiple peripheral neuritis seems to have been completely independent of the glycosuria, and was remarkable for its uncertain causation, and its very rapid and complete recovery.

J. D. ROLLESTON.

TREATMENT OF INFANTILE CEREBRAL DIPLEGIA. E. W. (133) SCRIPTURE, *Brit. Med. Journ.*, 1917, March 17, p. 363.

THE fundamental fact of this disease is the spasticity of the muscle. The act of attention is followed by excessive contractions that produce the familiar staring mask-like appearance. In walking, moving the arms, or in speaking, every muscle is thrown into violent contraction. Up to the present time all the methods designed to teach these people to walk and control their arms have consisted in exercises in which they are taught to try hard to control the jerkiness of the movements. By the new method the patient is trained to speak and act gently, as if he did not care to make any effort at all. The result is a weaker muscular action. In every act the patient carefully refrains from any mental effort, and the spastic muscles are aroused only by weak impulses. The patient learns to walk and speak by special training in graceful, easy, and relaxed movements.

INSCRIPTIONS OF SPEECH IN CEREBRAL DIPLEGIA, WITH (134) INDICATIONS OF A NEW METHOD OF TREATMENT. E. W. SCRIPTURE, *Proc. Roy. Soc. of Med.*, 1917, x. (Sect. for Study Dis. in Children), pp. 36-48.

INSCRIPTIONS of speech by patients with this disease show characteristic abnormalities. The essential abnormality is the bellowy, over-enunciated speech with monotony of tone. This arises from hypertonia (spasticity) and atonia (efforts to correct spasticity.) The only successful way of combating this is to diminish the will-impulses of the patient by means of training him to speak in a negligent don't care fashion, because his ordinary will-impulses are followed by excessive action of the spastic muscles. In a similar way this principle of training in a mental attitude of relaxation is applied to the movements

of the arms and legs, as in writing, walking, dancing, &c. The usual method of training by exercises in voluntary co-ordination is detrimental.

RECORDS OF SPEECH IN DISSEMINATED SCLEROSIS. E. W. (135) SCRIPTURE, *Brain*, 1916, xxxix., p. 455.

INSCRIPTIONS of speech by over twenty cases of disseminated sclerosis were made on a recording apparatus. In every one of them a peculiar abnormality in the vibrations was found, even in cases having apparently no speech defect. This abnormality was shown to be a registration of laryngeal ataxia. It occurs in none of the diseases usually confused with disseminated sclerosis. The presence of this abnormality can be considered as an actual physical proof of the presence of this disease just as a Wassermann is a proof of the presence of syphilis. The entire process of registration is purely mechanical, and there is no room for doubt. The sign is simply present or absent, and the conclusion is automatically positive or negative. Inscriptions were made of all the forms of speech found in this disease. In spite of their great variability they could all be reduced to the four principles of ataxia, anataxia (efforts to correct ataxia), hypertonia (spasticity), and anatonía (efforts to correct spasticity). The usual terms "staccato speech, scanning speech," &c., were shown to be misleading or erroneous.

SPEECH WITHOUT USING THE LARYNX. E. W. SCRIPTURE, (136) *Journ. of Physiol.*, 1916, l., Dec. 15.

THE patient, a girl, 17 years old, had worn a tracheotomy tube since the age of 3. No air passed through the larynx. She was nevertheless able to speak in a faint voice by collecting air in her pharynx. A faint tone was produced by vibration of the back of the tongue against the palate. A kind of pseudo-glottis was thus formed. Inscriptions of her speech showed various peculiarities.

SEVERE PARALYSIS OF THE SCIATIC NERVE FOLLOWING (137) GLUTEAL INJECTIONS OF QUININE. (*Paralysies graves du nerf sciatique consécutives à des injections fessières de quinine.*) J. A. SICARD, L. RIMBAUD, and H. ROGER, *Paris Méd.*, 1917, vii., p. 10.

THE writers have recently seen fifteen cases, of which they record six in detail, in which, owing to the defective technique, intra-gluteal injection of quinine for malaria was followed by severe pain and almost complete paralysis of the lower limb.

The sciatic nerve may be injured either directly by contact with the quinine solution, or indirectly by inflammation of the adjacent cellular tissue.

To avoid these accidents the writers recommend that the gluteal injection should be made in strict conformity with the rules laid down in the text-books, or that the injection should be given in the outer side of the thigh. J. D. ROLLESTON.

THE EFFECTS OF CINEMATOGRAH DISPLAYS UPON THE EYES OF CHILDREN. N. BISHOP HARMAN, *Brit. Med. Journ.*, 1917, i., Feb. 17, p. 219.

THE unpleasant effects associated with the cinematograph exhibition, so far as they affect the eyes, are due to the following conditions—(1) glare; (2) flicker; (3) rapidity of motion; (4) concentration of attention; and (5) duration of exhibition. These are discussed here, and it is considered that the best protection for the child will be secured by the following provisions—(1) the reasonable illumination of all parts of the hall not directly beside the screen; (2) the improvement of the movement of the film so as to reduce flicker, and the withdrawal of films immediately they are damaged; (3) an improvement in taking the picture so as to bring the rate of motion of the objects depicted more clearly to the natural; (4) the increase in the number of intervals in the show, and the interposition of exhibitions other than that of the optical lantern; (5) the limitation of shows for children to one hour, and the prohibition of "repeats"; (6) the reservation of the children's seats to the "optimum" position in the hall. Under such conditions, one show a week should do no harm to the eyes of a normal child. A. NINIAN BRUCE.

QUININE AMAUROSIS, WITH REPORT OF A CASE. ARTHUR J. (139) BALLANTYNE, *Brit. Journ. Ophthalmology*, 1917, March 1, pp. 153-160.

IN quinine poisoning complete loss of vision may be found in association with a normal condition of the fundus oculi, and a striking recovery of vision may take place in spite of the presence of well-marked fundus changes.

In all, or nearly all cases of quinine amaurosis, ophthalmoscopic changes, such as congestion of optic nerves and retina, pallor of the disc, narrowness of the retinal vessels, and cloudy opacity of the retina, make their appearance sooner or later, but there is no correspondence between the character or severity of

these changes and the intensity of the visual defect. The visual defect cannot, therefore, be due to such changes, but rather to a condition of the retinal elements invisible with the ophthalmoscope.

This change may be induced or aggravated, in the first place, by ischaemia, due to contraction of the vessels of the optic nerve and retina, but it is, in the main, the result of a direct toxic action of quinine upon the retina itself, and the ultimate recovery of central vision, with loss of peripheral vision, and failure of vision in twilight, suggests a selective action of the poison upon the rods.

A. NINIAN BRUCE.

THE INFLUENCE OF VASCULAR DISEASE IN THE RETINA
(140) **ON THE PROGNOSIS AS REGARDS LIFE.** P. H. ADAMS,
Brit. Journ. Ophthalmol., 1917, i., March, pp. 161-165.

RECORDS of 156 cases are given, and it is concluded that retinal lesions are considerably more common in women than men, and that not altogether due to child-bearing, as 38 of the 96 women were, as far as could be discovered, unmarried. The cases were most numerous between the ages of 60 and 70, and next between 50 and 60.

The older the patient the better the prognosis as regards life, irrespective of the presence of albumen in the urine to a large extent, whilst the younger the patient the worse the prognosis, especially so if albumen is present in the urine.

A. NINIAN BRUCE.

A PSYCHOLOGICAL ANALYSIS OF STUTTERING. WALTER B.
(141) SWIFT, *Journ. of Abnorm. Psychol.*, 1915, Oct.-Nov.

IN very brief form a complete, automatic, visualisation process may be developed by vocal drill in cases where previously there was total absence of all these visualisation processes during speech.

In relation to the stuttering, the symptoms disappeared in proportion as the picturing processes developed. This may therefore be considered as the "new treatment indicated" in this article. I next plan to present a long series of such cases so treated, presenting both the original absence of picturing and its final development. By that time I think I will be ready to give out my system of treatment in final form.

AUTHOR'S ABSTRACT.

. PSYCHIATRY.

OPTIMISM AND PESSIMISM. HENRY MAUDSLEY, *Journ. Ment. Sci.*, 1917, lxiii., Jan., p. 1.

THIS essay does not lend itself to abstraction. It is written with all the erudition and the delightful phraseology so characteristic of the author.

H. DE M. ALEXANDER.

THE ORIGIN OF MENTAL POWER. CASPER L. REDFIELD, *Journ. Ment. Sci.*, 1917, lxiii., Jan., p. 56.

ORDINARY biology teaches us that the inheritance of the child is not affected by the education of the parent. Men have been educated in the schools, horses on the race-track, and hunting dogs in the fields for generations, and investigation here tells a very different story from that of the biologists. It tells us that improvement in mental and physical power from generation to generation comes directly from educating each generation in succession, and informs us that whenever education in any generation falls below a certain minimum amount, then the next generation will decline in its inherited powers of that particular characteristic which lacked education.

Only older persons have acquired much effects from long-continued education. We have, therefore, in the age of the parents at the time of reproducing, a very definite test of the matter. If the effects of education are transmitted from father to son, then great men must come from parents, grandparents, and great-grandparents who were above the average age of parents when the average child is born. The average father is thirty-two years of age when the average child is born. The age of the father is the "birth-rank" of the child. From the investigation of the ancestries of 860 eminent men it was found the standard age of the father was forty years. A comparison was drawn between the birth-ranks of these eminent men and the birth-ranks of an average community, and it was found that each increase in the age of the father at birth of the son increased the son's chances of becoming eminent.

Those men who become eminent because of inherited opportunities, or because of some spectacular achievement, gravitate toward that end of the scale represented by the younger fathers; while those who become eminent by reason of pure intellectual power gravitate toward the end of the scale represented by the older fathers.

Ignoring idiots and the insane, sub-normal and feeble-minded men form the other end of the scale. When we test these cases

we find that they originate in children produced by uneducated parents who were usually less than twenty when those children were born, and these families are continued in their sub-normality by continuous generations of young and uneducated parents.

It is not necessary to sterilise, segregate, or transport the sub-normal in any effort to improve the race. This is futile. All that is necessary is to force education upon individuals to whatever extent possible, and prevent them marrying until they have reached their majority. Two generations of that proceeding will raise the sub-normal stock to normality.

H. DE M. ALEXANDER.

NOTES ON MENTAL DEFECT IN CRIMINALS. Sir BRYAN (144) DONKIN, *Journ. Ment. Sci.*, 1917, lxiii., Jan., p. 16.

THE first part of this paper criticises the recently increasing output of literature on "Criminology" which appears in various books, magazines, novels, &c., and much of which is borrowed, with no knowledge of the subject, from what are believed to be "scientific" authorities, and thus has its effects on the general public, and even on some criminals.

The second part considers the relation of mental defect to crime and criminals, and the author believes that the large majority of criminals convicted of most kinds of crime could not with any plausibility be dealt with either as being the subjects of defective intellectual capacity, or even of "mental deficiency" in the proper sense of that term. But there is a very notable minority of criminals of many kinds whose degree of mental defect not only, or always, of generally defective intelligence, is so manifest that for practical purposes they should be regarded and treated more or less similarly to the insane. They exhibit by their conduct, apart from the crime for which they have been convicted, highly probable evidence of their being the subjects of such defect of mind and brain as to render it fairly certain that the defect is congenital, and has but a very subordinate dependence on the "force of circumstances." Many of these cases bear a close resemblance to others occurring among non-criminals, and in all social grades.

Finally, a case of wilful murder is referred to in which the youth convicted of the crime—the plea of insanity having failed—could not have been certified as a "lunatic," nor probably accepted by the Board of Control as a proper subject for treatment in a State Institution for "mental defectives," nor certifiable either as a "person of unsound mind," or an "idiot," although he was, in the untechnical sense of these words, as certainly one as he was not the other. He was with equal certainty a person

"dangerous to the community," and the author considered him properly certifiable under the Mental Deficiency Act as a "moral imbecile," not only on account of the circumstances of his crime, but also from his attitude towards his own case, and of the history of his general conduct throughout his life prior to the crime.

H. DE M. ALEXANDER.

GENERAL PARALYSIS IN A CASE OF OXYCEPHALY. (Paralysie (145) *générale chez une oxycéphalique.*) H. FLOURNOY, *Nouv. Icon. de la Salpêtrière*, 1916-17, No. 1, p. 15.

OPTIC nerve atrophy in oxycephaly is not the result of narrowing of the fissures through which the nerves pass, but is secondary to increased intracranial and intraventricular pressure. Similarly third nerve paralysis is due to raised intraventricular pressure. In the course of oxycephaly sudden ventricular stasis is apt to arise, owing, perhaps, to the abnormal inclination of the aqueduct of Sylvius.

S. A. K. WILSON.

DEMONSTRATION OF SPEECH INSCRIPTIONS FROM A CASE (146) OF JUVENILE GENERAL PARALYSIS, WITH HYPO-PITUITARISM. E. W. SCRIPTURE, *Proc. of Roy. Soc. of Med.*, 1916, x. (Sect. for Study Dis. in Children), pp. 10-13.

THE case recorded is that of a boy where there was doubt concerning the diagnosis. Inscriptions of speech showed the same abnormality as that found in early general paralysis, namely, asaphia (*v. Review*, 1917, xv., p. 35). They also showed indications of apraxia and of bulbar paralysis. The diagnosis was thus fixed as juvenile general paralysis.

VARIATIONS IN THE SENSORY THRESHOLD FOR FARADIC (147) STIMULATION IN PSYCHOPATHIC SUBJECTS.—III. THE DEMENTIA PRÆCOX GROUP. G. P. GRABFIELD, *Boston Med. and Surg. Journ.*, 1915, clxxiii., Aug. 5, pp. 202-205.

THE determination of the sensory threshold for faradism shows that pathological values are constantly obtained in only about one-third of the cases of dementia præcox, and hence that this determination is of diagnostic value only in this percentage of cases.

The mechanism governing emotional tone in dementia præcox appears by this test to be different from that which governs this phenomenon in manic-depressive insanity (*v. Review*, 1916, xiv., p. 188).

The correlation of catatonic symptoms with high threshold values parallels the correlation between these symptoms and post-Rolandic cortical lesions. From this it is probable that the "arrival platforms" for faradic stimuli lie in the post-Rolandic region.

The correlation between somatic delusions and high threshold values is evidence in favour of the suggestion that these delusions are associated with a perversion of the receptor mechanism.

A. NINIAN BRUCE.

CRIME IN DEMENTIA PRÆCOX. RALPH M. TOLEDO, *Journ. Ment. Sci.*, 1917, lxxiii, Jan., p. 100.

FOUR cases are cited in which a violent crime—the result of a faulty process of reasoning and with a certain amount of pre-meditation—was committed in the pre-demented stage of dementia præcox. Two examples are given of crime of a fraudulent nature committed by two men, hitherto of good character, as the result of contracting friendships with bad characters, and thus illustrating the abnormal facility present in the predominal stage of the disease. All six cases rapidly exhibited the ordinary symptoms of dementia præcox after conviction. H. DE M. ALEXANDER.

RESULTS PRODUCED IN DEMENTIA PRÆCOX OR SO-CALLED (149) "ENDOGENOUS DEMENTIA" BY THE INFUSION OF SODIUM CHLORIDE SOLUTION. N. ISHIDA, *Amer. Journ. Insan.*, 1917, lxxiii, Jan., p. 541.

IN ten cases of dementia præcox intravenous or subcutaneous injections of 0.9 per cent. of common salt solution were given. The cases are reported very summarily, but the author feels that his results were encouraging, and in 50 per cent. of cases there seemed an "awakening of interest in work."

D. K. HENDERSON.

PSYCHIATRIC FAMILY STUDIES. A. MYERSON, *Amer. Journ. Insan.*, 1917, lxxiii, Jan., p. 355.

THIS paper of 130 pages is a most valuable contribution to the study of heredity in relation to mental disease. The literature on the transmutation of the psychoses and of the family studies done by others is briefly reviewed. The author then offers a criticism of the work of Koller and Diem on the insane and non-insane. Koller and Diem found that the insane are slightly more tainted, if all degrees of taint in all relatives (excepting children)

are considered. They are far more tainted by insanity, and more especially by insanity in the parents, and through direct inheritance by character anomalies and by alcohol. The sane have more nervous diseases, apoplexy, senile dementia in their direct and collateral heredity, and generally are more heavily tainted through grandparents and collaterals than are the insane. Koller and Diem's statistics are, however, vitiated by the fact that the eccentric, psychopathic, and peculiar are considered among the normal, and secondly, in the majority of cases, the normal were under 30 years, whereas the insane ages ranged much higher.

In considering the marriage rate of the insane, Myerson has compared statistics taken from several hundred cases, male and female, from the alcoholic psychoses, general paresis, dementia præcox, and senile psychoses. It was found that the males in the alcoholic, parietic, and dementia præcox groups marry less than do the females. In the senile cases, though the percentage of married men is greater, the totals of those who have married at one time or another are about equal. The striking differences are really seen in the dementia præcox group, where the married males were just about half as many as the married females, but the married females in this group were very definitely fewer than in either the alcoholic or parietic groups. Dementia præcox, therefore, would seem to operate against self-perpetuation.

Of the 22,300 patients admitted to the Taunton State Hospital from 1854 to 1916, there were 1,547 related to one another, and these represented 663 families. There was 1 four generation family, 23 three generation families, 333 two generation families, and 307 one generation families. The analysis of the relationship of the members of these groups closely corresponded to Mott's figures from the London County Asylums. The mother-son relationship is much less frequent than mother-daughter (as 55 is to 80), but the father-son relationship is only slightly less common than the father-daughter (as 55 is to 59). The author's statistics also corroborate Mott's figures in regard to "Anticipation or Antedating," but the fact that the figures have practically all been collected during a period of thirty years invalidates one drawing any definite conclusions.

Detailed case histories are given of all the cases wherein three or more generations are represented, and a part of the cases in which two generations are represented. In analysing these records the following main conclusions are arrived at:—

Paranoid states in the immediate ancestors are followed either by dementia præcox or paranoid states in the descendants. Dementia præcox breeds dementia præcox; epilepsy, moral imbecility, or feeble-mindedness may appear. Clean-cut cases of manic-depressive insanity are usually followed by manic-depres-

sives, but dementia præcox may also appear. Idiocy, feeble-mindedness, &c., follow much less closely on manic-depressive than on dementia præcox.

In the involution psychoses the great majority of the descendants belonged to the dementia præcox groups. Senile psychoses are usually followed by dementia præcox.

Neither in regard to organic brain disease nor alcoholic psychoses can anything very definite be said.

"It will thus be seen that all roads lead to dementia præcox, and from thence to imbecility."

D. K. HENDERSON.

THE KORSAKOFF SYNDROME (TOXÆMIC CEREBROPATHY)
(151) **IN PREGNANCY.** EUGENE RIGGS, *Amer. Journ. Insan.*, 1917, lxxiii, Jan., p. 525.

THE case is described of a young woman, 25 years old, who, in the second month of her second pregnancy, developed pernicious vomiting. Symptoms suggestive of appendicitis arose, her appendix was removed, and her symptoms were immediately alleviated. Three weeks later she suddenly developed a retrobulbar neuritis, albumin and indican appeared in the urine, she became stuporous, and could not be roused to answer questions. The uterus was cleaned out, and a healthy foetus removed. Following this the patient showed a delirious, confabulatory state, her knee and Achilles jerks were absent, the nerve trunks were sensitive to pressure, and the vagus was also involved. Seven months later, although still weak, she had practically regained her normal condition.

A second case is also reported occurring in an alcoholic. A short review of the literature is given.

D. K. HENDERSON.

PROLONGED NASAL FEEDING. E. E. MOORE, *Lancet*, 1917, i, (152) April 21, p. 635.

AN insane man, dying at the age of 81 years, had been fed three times daily through the nasal tube for twenty-one years seven months prior to his death. During this period his total daily diet consisted of 4 pints of milk, 2 pints of beef tea or beef extract, 3 eggs, 2 potatoes, and 2½ ounces of sugar, and he had never been seen to drink even water.

H. DE M. ALEXANDER.

**CONSANGUINITY AMONG PATIENTS AT THE NEWBERRY
(153) STATE HOSPITAL, MICHIGAN. M. P. KEMP, *Amer. Journ.
Insan.*, 1917, lxxiii., Jan., p. 487.**

AMONG 400 women patients in the Newberry State Hospital, fifty had relatives under treatment. This group has been examined in regard to the exact relationship with the following results:—

Mother and daughter	-	-	-	-	8
Mother and son	-	-	-	-	3
Father and daughter	-	-	-	-	7
Brother and sister	-	-	-	-	9
Sisters	-	-	-	-	4
Aunt and niece	-	-	-	-	6
Cousins, nieces, &c.	-	-	-	-	13
					—
					50

No conclusions are drawn in regard to the clinical forms of the mental disorder.
D. K. HENDERSON.

**A HISTOLOGICAL STUDY OF THE OPTIC NERVES IN A
(154) RANDOM SERIES OF INSANE HOSPITAL CASES. MYRTLE
M. CANAVAN, *Journ. Nerv. and Ment. Dis.*, 1916, xliii., March, pp.
217-230.**

FORTY cases, or 68 per cent., of a random series of 58 cases of mental disease autopsied at the Boston State Hospital showed obvious and important chronic changes in one or both optic nerves (one, 13; both, 27).

In the same series of 58 there were but 34 which showed chronic spinal cord changes by the same method (Weigert myelin sheath).

There were 7 cases which showed very slight changes in the spinal cord (although in all instances definite changes) when there were no changes demonstrable in the optic nerves.

Of 18 syphilitic cases (clinical evidence in some cases supported by the Wassermann reaction) there were 15 showing optic nerve changes—one eye, 3; both eyes, 12.

In one case a spirochæte was demonstrated by the Levaditi method in the pial sheath of the optic nerve in a case diagnosed general paresis (although possibly one of cerebro-spinal syphilis).

A. NINIAN BRUCE.

PSYCHOANALYSIS: A NEW PSYCHOSIS. (*Une psychose nouvelle: (155) la psychoanalyse.*) YVES DELAGE, *Mercur de France*, 1916, 1st Sept.; translated by T. DRAPES, *Journ. Ment. Sci.*, 1917, lxxiii., Jan., p. 61.

THIS is a clever, amusing, and at the same time scathing skit on psychoanalysis as preached by Freud and his pupils. The author rightly considers that he "has not exceeded the limits of just criticism, and that the apparent exaggerations in this satire are, even as regards details, in exact correspondence with the enormities of the theory."

H. DE M. ALEXANDER.

CHRONIC INFECTIONS BY THE BACILLUS OF INFLUENZA (156) AND THEIR IMPORTANCE AS CAUSES OF NERVOUS DISORDERS. W. FORD ROBERTSON, *Journ. Ment. Sci.*, 1917, lxxiii., Jan., p. 89.

CHRONIC infection by the bacillus of influenza is a common cause of ill-health, though as yet hardly recognised: the nervous symptoms—asthma, mental depression, neurasthenic phenomena, insomnia, headache, "gripping sensations" in the head, neuralgic or rheumatic pains—that occur in some of the cases among the general population are so severe that it seems probable that, in persons with a hereditary predisposition to insanity, grave mental disturbances may easily be induced. The bacillus of influenza, when acting as a chronic infecting agent, never occurs alone, but is always accompanied by other pathogenic bacteria, thus influencing the clinical picture. Immunisation with sensitised vaccines has been, in the author's hands, uniformly successful in eradicating the symptoms, but it is necessary to treat in a similar way one or more accompanying infections. (Notes of cases presenting special features are appended.)

H. DE M. ALEXANDER.

REMARKS ON STATE CHARITIES' LAWS WITH SUGGESTIONS (157) FOR A STANDARD TYPE TO COVER THE NEEDS OF PRESENT-DAY MANAGEMENT, AND ALSO THE MENTAL HYGIENE MOVEMENT, LOOKING TO PREVENTION. SIDNEY D. WILGUS, *Amer. Journ. Insan.*, 1917, lxxiii., Jan., p. 499.

THE author postulates the belief that practically all forms of mental disease are due to a faulty ancestry, and the problem to be met is to alleviate and treat mental disorders which have already arisen, to investigate the causes of these abnormal conditions, and the means of preventing their development. The author then discusses in detail the different methods at present in force for dealing with this problem in New York State, in Illinois, and in Tennessee.

D. K. HENDERSON.

Reviews

THE BASLE ANATOMICAL NOMENCLATURE [BNA], being an
(158) **alphabetical list of terms showing the old terminology, the
B.N.A. terminology, and the suggested English equivalent.**
E. B. JAMIESON, M.D. Pp. viii+91. W. Green & Son, Ltd.,
London and Edinburgh. 1916. Pr. 6s. net.

THE list of names comprised in the revised anatomical nomenclature was prepared, after six years' work, by an international commission of anatomists, and was submitted to the meeting of the Anatomical Society at Basle in 1895, where it was adopted by the Society—hence the name Basle Nomina Anatomica (B.N.A.).

The need for a revision of the nomenclature had long been felt, as different nomenclatures had grown up in different countries and much confusion had resulted in consequence. Latin was the language adopted for this nomenclature as it is common to all countries. It is astonishing that such a universal change as this should have met with so little opposition, and that so many clinical teachers have seen their way to adopt it. The scheme is criticised here by Dr Jamieson in his introduction in a most able manner and a number of inconsistencies and errors pointed out, *e.g.*, the name of the *appendix vermiformis* has been changed to *processus vermiformis*, but the artery has remained the *arteria appendicularis*. No name, however, has been more criticised than *radial* nerve in place of *musculo-spiral* nerve, as hitherto, in English, it has been applied to another nerve, one of the terminal branches of the musculo-spiral. This change makes a harmonious arrangement of nerves in the arm—radial, median, and ulnar—and it is only to be regretted that like the median and ulnar, the name does not hold sway from the axilla to the hand, and that another name was not devised for its deep branch.

Ventralis, *dorsalis*, *cranialis*, and *caudalis* replace *anterior*, *posterior*, *superior*, and *inferior* respectively, although the latter terms are retained for use in the head and pelvis, but the Commissioners seem to have realised that too rigid adherence to the former terms would make lucid passages appear confused, as the student would almost certainly think of a given structure being "in front of" another or "anterior" to it.

An alphabetical list of the English names is placed in the first column, the Basle names in the second column, and a translation or an equivalent in the third. These equivalents are mere suggestions, as there is as yet no authoritative English list based on the Basle Nomina Anatomica. Those given here are for the

most part translations and correspond fairly closely with those which have been employed in the English text-books which have adopted the B.N.A. This list ought to prove of great use.

THE SECRETION OF URINE. ARTHUR R. CUSHNY, M.D., LL.D., (159) F.R.S. Pp. xii+241, with 36 figs. Longmans, Green, & Co., London. 1917. Pr. 9s. net.

THIS is the second of the series of monographs on physiology published under the editorship of Prof. Starling, and intended to set out the progress of physiology in those chapters in which the forward movement is most pronounced. The first monograph of the series by Dr Gaskell upon the involuntary nervous system has already been reviewed (*v. Review*, 1916, xiv., p. 325).

It would be difficult to find a more suitable subject for discussion than the function of the kidneys, as no other organ in the body has suffered so much from poor work, and our reliable knowledge of the secretion of the urine is badly in need of restatement.

It is often complained that the physiology of the kidney is made up of a wrangle between the two great views of its activity; but, on the other hand, the many isolated observations need to be correlated with each other. The first theory is that of Bowman-Heidenhain, which amounts to little more than a statement that the kidney secretes the urine by the vital activity of its cells. Such a view has the advantage that no possible conjunction of conditions can be imagined which cannot be attributed to some special activity of unspecified cells, whose activity is governed by no known laws, and whose anatomical position in the organ can be arranged to suit the circumstances. It is impregnable as a defensive position, but it offers no point from which advance may be made. Two years after Bowman's view was published, Ludwig (1814) put forward another theory of the secretion of the urine. He held that the capsule was a simple filter, which allowed all the constituents of the plasma to pass through it except the proteins, and that in the tubules this filtrate was elaborated into the urine by the return of much of the fluid into the blood through a process of diffusion. Until Heidenhain developed Bowman's view, the ideas of Ludwig seem to have prevailed, but after that masterly piece of work appeared, they suffered an eclipse. More recently the advances in physical chemistry of the last twenty-five years have finally disproved Ludwig's view by showing that the known physical forces are inadequate to form from the blood plasma a fluid of greater osmotic pressure. From these there gradually evolved what Prof. Cushny calls the

"modern theory," which accepts the general scheme of filtration and reabsorption of Ludwig, but, appreciating the inadequacy of the known physical forces, supplements them as far as is necessary by the "vital activity" postulated by Heidenhain. The secretion of the urine is to be considered as consisting of two distinct processes, differing not only in site but also in nature. The first of these, the filtration, occurs in the glomerulus, and is purely physical; the second, the reabsorption, occurs in the tubules, and depends on the vital activity of the epithelium.

The whole question of kidney function is discussed here in such a masterly manner that this cannot fail to be the work which all future workers will regard as the foundation upon which they may build. It has collected and summarised our present knowledge, and criticises in an unusually sane manner the numerous theories advanced to explain renal activity. The bibliography is apparently complete.

RHINOLOGY: A TEXTBOOK OF DISEASES OF THE NOSE AND (160) THE NASAL ACCESSORY SINUSES. PATRICK WATSON WILLIAMS, M.D. Pp. xvi+273, with 47 plates. Longmans, Green, & Co., London. Pr. 12s. 6d. net.

THE reader will find here an excellent text-book presenting the science as well as the practice of rhinology. It opens with a description of the methods of examining the nasal passages; this is followed by a short account of the anatomy and physiology of the nose, after which the different clinical conditions are described in turn. Special attention has been given to the clinical anatomy of the nose and the variations in the different accessory sinuses. One chapter is devoted to the nasal neuroses. Reflex nasal neuroses most frequently excite physiological reflexes and other symptoms in the upper respiratory tract, *e.g.*, sneezing, coryza, and vascular turgescence; next in frequency come morbid reflex phenomena in the lower tract, *e.g.*, asthma, vasomotor bronchitis, &c., while only very rarely are epilepsy, melancholia, cardiac symptoms, &c., dependent on nasal sources. Spontaneous discharge of cerebrospinal fluid through the nose, termed *cerebrospinal rhinorrhœa* by St Clair Thomson, is a rare condition. Apparently the fluid escapes through a small hole in the dura mater by the side of the crista galli, or through the cribriform plate of the ethmoid. Generally there is a history of headache or other mental symptoms, which are relieved with the escape of the cerebrospinal fluid. The author had a case of this condition which persisted for several years and became normal after repeated lumbar punctures.

Hay fever is simply paroxysmal sneezing set up by particular

forms of irritating dust, viz., pollen grains. Attention was first seriously directed to the condition by Bostock in 1819. While the exciting causes are universal and the predisposition very common, the affection itself is relatively rare. The third factor which is thus generally necessary is some morbid condition or abnormality of the nasal passages, such as (1) hypertrophic rhinitis, (2) spurs or bony projections of the turbinals or septum, (3) septal deviations, (4) polypi and adenoid hypertrophy of the nasopharynx, and (5) peculiarly sensitive areas. Cocaine should never be applied locally for relief, as it only tends to aggravate the condition after its transient good effects have passed off.

The large percentage of patients with large nasal polypi who do not suffer from asthma, and the very large percentage of true asthmatics in whom no nasal polypi can be found, tend to prove fairly conclusively that there is no direct connection between nasal polypi and asthma as cause and effect. The nasal abnormalities which are frequently associated with asthma are causes of intranasal excitation and similar conditions resulting from infective inflammations in the accessory cavities, which, while resulting often in polypi, are also efficient peripheral causes of asthma by irritating the bulbar respiratory centres and through them the efferent nerves to the bronchioles.

A large number of useful prescriptions are given in an appendix, together with an account of the best methods of examining the nose and nasal sinuses post mortem. There is an extensive bibliography. One of the special features of the book, however, is the large number of illustrations in the text. Special reference must, however, be made to a series of twenty-seven stereoscopic plates illustrating numerous different pathological and other nasal conditions. These are of very great beauty and have been most carefully prepared and reproduced. They add greatly to the value of the book.

THE NEUROTIC CONSTITUTION. OUTLINES OF A COMPARATIVE INDIVIDUALISTIC PSYCHOLOGY AND PSYCHOTHERAPY. ALFRED ADLER. Authorised English translation by Bernard Glueck, M.D., and John E. Lind, M.D., with an introduction by Dr William A. White. Pp. xxiii+456. Moffat, Yard & Company, New York. 1917. Pr. \$3.00 net.

THE study of the neurotic character is an essential part of neuropsychology; it is here presented to us from a new point of view. The neurotic shows a series of sharply emphasized traits of character which exceed the normal standard, such as marked sensitiveness, irritable debility, suggestibility, egotism, estrange-

ment from reality, and often more special traits such as tyranny, malevolence, a self-sacrificing virtue, coquetry, anxiety, and absent-mindedness.

Adler considers that the basis of these neurotic character traits is to be found in the fact that the person showing such is the possessor of what he terms "an inferior organ," and that in consequence of the feeling of inferiority which this produces, an effort is made by him to compensate for this by so ordering his life and so regulating his every act that he may find that security of which the feeling of inferiority has robbed him. This is the fictitious goal of the neurotic, and is also the fundamental and ultimate cause of the symptoms which develop when he is no longer able to succeed in dealing thus with reality. The point of view is thus rather from the organic than from the functional side, and forms a connecting link between the pathologist and the psychologist. Adler here works out at considerable length the psychological characteristics of persons who have had demonstrably inferior organs, either clinically evident or discovered at autopsy. From this he believes he has been able to show that the predominant traits of character are the result of an effort on the part of the individual to overcome a feeling of inferiority resulting from an inferior organ. He regards the neuroses and psychoses as constructive creations which are built up under the influence of a dominant guiding idea which collects and unites into a group those psychic elements which it can make use of in its effort to attain security. The attempt fails because the direction is false, the condition becomes unstable, and the neurosis is the practical result.

Adler considers that three of the fundamental views of Freud are erroneous, viz., first, that the libido is the motive force behind the phenomena of the neuroses; second, that the neuroses have a sexual etiology; and third, that the neurotic is under the influence of infantile wishes which come to life in dreams. He thinks that at the onset of the development of a neurosis there stands threateningly the feeling of uncertainty and inferiority, and that this demands insistently a guiding, assuring, and tranquillizing goal to make life bearable. As this leads to an exaggeration of the ego, the neurotic becomes conspicuous in society because of his evident inability to adapt himself, and the apperception of experiences by this type of character leads to special psychopathological features. None of the traits of character of the neurotic are essentially new, and he shows no single trait which cannot likewise be demonstrated in the healthy individual. It thus becomes necessary to establish an empiric basis of normality by which different grades of deviation can be compared, and thus a comparative individualistic psychology is built up.

The translators appear to have had considerable difficulty in finding English equivalents for many of the German words and expressions, but they have been quite successful in interpreting the meaning of the author.

FREUD'S THEORIES OF THE NEUROSES. EDUARD HITSCHMANN (162) (Vienna). Authorised translation by Charles Rockwell Payne, M.D. Introduction by Ernest Jones, M.D. Pp. xxiv + 257. Moffat, Yard & Company, New York. 1917. Pr. \$2.00 net.

THE lack of a systematic arrangement of Freud's writings has made it difficult for a beginner to find a suitable book to commence the study of his views and theories of the neuroses, and this work has been accordingly undertaken with the object of presenting a condensed account of these views, and of sketching, as far as possible, the framework upon which they are moulded. The constantly increasing extent of the publications dealing with these subjects, and the gradual elaboration which Freud has himself been responsible for has added considerably to the difficulty of a proper understanding, especially as a number of his colleagues have lately definitely separated themselves from him, and put forward different views of their own.

We have here what is meant to be purely a review of Freud's theories, and is intended to serve as an introduction to his work, and to supply those wishing to make a study of his views with a starting-point from which to lead off. It gives a brief description of the early work of Charcot, Janet, and Breuer which led up to Freud's later studies, and then discusses in turn his theory of the neuroses, the true neuroses, the sexual instinct, the unconscious, the dream, hysteria, the obsessional neuroses, psycho-analytic methods of investigation and treatment, general prophylaxis of the neuroses, and the applications of psycho-analysis. It concludes with a chronological review of Freud's publications and a list of those translated into English.

As a good interpretation of Freud's views, this may be safely recommended, and thus serves as a useful introduction to more detailed and special publications.

MENTAL NURSING. W. H. B. STODDART. Pp. 98. The Scientific (163) Press, Ltd., London. Pr. 2s. 6d. net.

THIS book consists of a number of chapters upon mental nursing which have all previously appeared as lectures in the *Nursing Mirror*, and which were written to give the prospective mental nurse an idea of the work he or she contemplated. It was also designed to furnish those actually engaged in mental nursing

with a practical guide in the management and care of the insane. The book fulfils its purpose well, the chapters being easy to follow and practical; and has the advantage of not being overloaded with details of the anatomy and physiology of the nervous system. Many useful hints and ideas may be obtained from it, especially with regard to details of nursing and the proper way to treat mental cases. It may thus be safely recommended to mental attendants.

HANDBOOK OF MASSAGE FOR BEGINNERS. L. L. DESPARD.
(164) Pp. xvi+247. Henry Frowde and Hodder & Stoughton, London.
1915. Pr. 6s. net.

At a time like the present when the need for massage is so great, especially in the increasingly large group of nervous cases produced by the war both at home and abroad, and when the necessity for a proper understanding of the particular conditions likely to benefit from such treatment requires to be so clearly understood, a book such as this is of special value. It is essentially a book for students of massage who intend making it a serious study, but the information is so simply and clearly given that it cannot but fail to prove most useful to the physician and surgeon, who in many cases seem to be curiously ignorant of the distinction between those conditions which obtain real benefit from such treatment, and those which do not.

Massage is here defined as "the scientific manual application of certain movements, such as effleurage, stroking, pétrissage, kneading and tapôtement to the human body, by which morbid conditions of the tissues are relieved," and it has to be noted that such treatment should only be carried out by persons who have undergone proper training.

The whole subject is here briefly brought under discussion, and the best methods of applying massage to joints and limbs, &c., for the correction of paralyses and deformities is described. A special chapter is devoted to massage in cases of bullet and shrapnel wounds, "frost bite" and traumatic neurasthenia. The last chapter deals with medical electricity, and there is a glossary defining most of the medical terms used in the book.

THE CITY OF DIN. A TIRADE AGAINST NOISE. DAN
(165) M'KENZIE, M.D. Pp. viii+115. Adlard & Son, London. 1916.

THIS book is a protest against all unnecessary noise. The author believes no reform in the world of to-day is at once so necessary and so easy to accomplish. He defines noise as unpleasant and painful sound. Thunder, the crow of the cock, the song of the

corn-crake, and such natural sounds are not to be considered as noise. But civilisation, at least modern civilisation, is noise; and the more it progresses the noisier it becomes. It is inferred that ancient cities were quiet because both the Athenians and the Romans practised the art of public speaking in the open air, and all their political gatherings were held in the streets and open places.

Noise produces exhaustion of the nerve centres; it also produces muscular tension and sustains it at an elevated level, as shown by the fatigue of a railway journey and the relief obtained by inserting ear-plugs. Provided the impulse be not too intense or too prolonged, and the brain not enfeebled by tiredness or disease, the nervous system can actually dampen or stifle an afferent impulse, probably by scattering its effects broadcast so as to destroy it for all practical purposes and thus render it harmless. Otherwise the nerve centres simply side-tract the impression and divert the stream of painful impressions into other subsidiary channels, which always lead to muscles. Rhythmic sounds lead naturally to rhythmic movement, as in marching to music, due largely to the fact that they to some extent relieve volition of the necessity for repeated effort by substituting for it a regular recurrent auditory stimulus to the rhythm of which the physiological timing of nerve and muscle movement seems naturally to adapt itself.

The author does not confine himself to the noises of ordinary life, but includes occupational noise-deafness from destruction of the auditory nervous apparatus from excessive stimulation, and numerous other noises, including the noise of battle. He emphasises the prime necessity of controlling the production of noise at its source if lasting good is to be done. The book is most readable.

HUMAN TEMPERAMENTS. Studies in Character. CHARLES (166) MERCIER, M.D., F.R.C.P. Pp. 91. The Scientific Press, Ltd., London. Pr. 1s. 3d. net.

THIS little book consists of thirteen short essays describing some common and well-known types of character as observed by Dr Mercier. It opens with a general description of temperament, and then proceeds to discuss in turn the artistic, religious, envious, jealous, and suspicious temperaments, the faddist, the practical man, the man of action, and the philosopher. The temperament of the artist is carefully distinguished from the artistic temperament, and the distinction between cleverness and capability is pointed out. The style is vivacious and racy, and many shrewd remarks are interpolated. No one interested in the study of human nature can fail to find much to interest as well as to amuse him here.

ACUTE POLIOMYELITIS, its nature and treatment. FREDERICK E. (167) BATTEN. Pp. 104, with 43 figs. 1916. John Bale, Sons & Danielsson, London.

THIS monograph represents the Lumleian Lectures for 1916, delivered at the Royal College of Physicians, London, last year, and has been already published in *Brain* (1916, xxxix.), and in abstract in the *Lancet* (1916, April 15).

It gives an excellent digest of our present knowledge of infantile paralysis from the points of view of epidemiology, pathology of the acute, chronic, and atrophic stages, experimental work, serum diagnosis, clinical manifestations, and treatment. The methods of making and applying celluloid splints are described in an appendix, and a bibliography of the literature under each of the above headings is given. The present attitude of the American and Continental schools is summarised briefly and clearly, and makes the book a useful work of reference. The illustrations are numerous and helpful.

The disease became compulsorily notifiable for London in September 1911, on the recommendation of the College of Physicians, and in September 1912 for the whole country. The value of this is clearly seen here. The study of the notified cases of poliomyelitis in England during the years 1912, 1913, 1914, and 1915 shows that the disease is distributed in this country in a very irregular manner, and is indicated here by several maps and diagrams.

SYPHILIS AND THE NERVOUS SYSTEM, for practitioners, neurologists, and syphilologists. MAX NONNE. Authorised translation from the second revised and enlarged German edition by Charles R. Ball, B.A., M.D. Pp. xxiv+450, with 98 illustrations. Second American edition revised. J. B. Lippincott Company, Philadelphia and London. Pr. 18s. net.

THE second American edition of this book is a translation from the third German edition published in 1915. The general arrangement of the material has not been greatly altered, except as far as was necessary to bring it up to date. This has affected mostly the chapters upon general paralysis and tabes, and the numerous recent contributions to serological reactions and therapy.

The book opens with a general account of the pathology and etiology of nervous syphilis and syphilitic endarteritis; this is followed by chapters upon cerebral and basal syphilitic meningitis with their symptomatology and prognosis, neuroses, and psychoses in syphilitics and in cerebral syphilis, general paralysis and syphilis, syphilis of the spinal cord, meningomyelitis, tabes, cerebro-spinal syphilis, peripheral nerve syphilis, and hereditary

syphilis of the nervous system. The last three chapters are devoted to the Wassermann reaction, prophylaxis and salvarsan therapy.

While nervous syphilis cannot be regarded as a relatively frequent nervous disease, it occurs about twice as often as cerebral tumour and disseminated sclerosis. Hjelmman states that cerebral syphilis develops in from 15 to 25 of every 1,000 cases of syphilis. It is possibly on the increase. It may develop in any stage of syphilis, and the tertiary stage is by no means the most frequent; Naunyn thinks that the first year after the infection is commonest, but it may be delayed for as long as forty years. Nonne considers that no matter how intelligent and energetic the treatment of the primary or secondary lesion may be, the possibility of a later nervous involvement cannot be eliminated. At the same time, the importance of thorough treatment of every primary and secondary case cannot be too strongly emphasised. Such cases may a few months later fail to disclose the slightest stigmata of syphilis. A large number of healthy children is no contradiction of past syphilis in the parents.

Primary and secondary syphilis may show a positive Wassermann reaction in the cerebro-spinal fluid without the patient manifesting any symptoms of nervous disease, but it has yet to be proved that these are the cases which later develop tabes, paresis, and cerebro-spinal syphilis. The absence of a Wassermann reaction in the blood does not exclude the possibility of syphilis; its presence "can tell us no more than that the individual has at one time been affected by syphilis." In tabes it is present in only 60 to 70 per cent. of cases. Its absence is only of differential diagnostic importance in general paralysis where it is positive without exception in over 95 per cent. of cases.

Considerable attention is paid to prophylaxis and treatment. Physicians usually are too optimistic in their prognosis of organic nervous affections recognised as syphilitic. Mercury and iodine are effective in all the diseased processes of syphilis, but while mercury acts only or only essentially upon the early processes, iodine influences chiefly the tertiary stage, and is specially recommended for certain early symptoms, such as occipital headache. Oppenheim gives the preference to mercury in syphilis of the nervous system. Neisser states that when administering mercury by injection, iodide should never be given at the same time, as the formation of local gummata may occur. "In by far the greater number of cases the superiority of salvarsan over mercury and iodide is not apparent." Optic atrophy is not a contraindication. The book closes with a bibliography of the literature and an index. This edition is a great improvement upon its predecessor.

Review

of

Neurology and Psychiatry

Original Articles

A CASE OF PATHOLOGICAL LYING OCCURRING IN A SOLDIER.

By D. K. HENDERSON, M D.,

Temporary Lieutenant, R.A.M.C., Lord Derby War Hospital, Warrington.

It is a well-known fact that, among the large group of cases which goes by the name of *high-grade mental deficiency*, lying constitutes a not unimportant symptom. Since a section of this hospital was opened for the reception of cases of nervous and mental illness, upwards of 1,400 patients have been received, of whom the various grades of mental deficiency have been richly represented. The case to be recorded, however, is to my knowledge the only one of its kind so far received, and it is presented not only on account of its dramatic interest, but principally on account of its bringing up important educational, sociological, and administrative problems.

No. 27,369, a private, attached to the 15th Battalion Durham Light Infantry, was, on 14th October 1916, admitted to the Lord Derby War Hospital from Netley.

On 11th September 1916 he had been admitted to No. 3 General Hospital, France, in a noisy, excited, insolent state, and was found to be not at all amenable to discipline. He said that he saw the spirits of dead people, that he heard them talking to him, and that it seemed principally to be his sister's voice urging him to lead a better life. Early in October 1916, when admitted to Netley, he said that he had always been a spiritualist, and had

been in the habit of conversing with dead people. He stated that he was a Frenchman, that owing to a quarrel with his parents he had gone to sea, and in 1908 had enlisted at Bristol in the British Army; that since that time he had been with the colours, and on 12th August 1914 he had gone to France with his regiment. In addition he stated that in September 1915 he had been wounded in the arm at Loos, but in February 1916 returned to the front line, and on 1st June 1916 had been "shell-shocked." Following this he lost himself, and did not know where he was until on 22nd July 1916 he was arrested as a deserter, was then sent to the 65th Field Ambulance, and later to No. 3 General Hospital.

On admission to this hospital (14th October 1916) he was quiet, orderly, co-operated in an examination, and gave no trouble. He considered himself quite well, said that he felt perfectly happy, and requested to be sent back to his regiment. He now gave the following account of his military career and life in general. He stated, as formerly, that he had enlisted in the British Army in 1908, and that he had gone to France in August 1914, and about February 1915 had received a flesh wound in the arm and an injury to the small toe of his right foot at Neuve Chapelle. On his recovery he was attached to the 15th Durham Light Infantry, and on 22nd July 1916 (formerly said 1st June) was blown up by a shell explosion, and following this remembered nothing until 5th August, when he found himself in an hospital in Boulogne. He was then sent back to his regiment, but about a month later was suddenly missing, as he states that he went off to pay back an old score on a former comrade in the Gloucesters who had at one time insulted his sister. He explained that he must have lost himself, as some days later he was arrested by the military police, and put under observation in the 65th Field Ambulance.

The above story was given without faltering, he showed no special emotional disorder, and no hallucinations or delusions could be elicited. He maintained, as he had done at Netley, that he was a spiritualist, said that following the shell explosion his head had been in a whirl, he had suffered from insomnia, and had seemed to hear his sister's voice. His memory seemed to be excellent, he did simple calculations correctly and quickly, and had an excellent grip on school knowledge.

In regard to his personal history, he said that he had been born

near Marseilles, and that he was 30 years old. His father had been a Spaniard of independent means, his mother was a French-woman, and he himself had been educated and brought up with the idea of his entering the French Army. With this in view he had been educated at a military academy at Paris, but, while there, had fallen into bad company, adopted wicked ways, and in consequence of his drunkenness had quarrelled with his parents and ran away to sea. Previous to his coming to England in 1908, however, he had returned to France, had married, but on account of his drunken habits had been divorced.

Physically.—He was a small, well-nourished, attractive-looking man, who looked younger than his alleged years, and perhaps might be described as rather effeminate looking. He showed no disease of his organs, and there were no neurological signs.

The above story corresponded in the main so well with the account given at Netley, that no special suspicion was aroused by it, and any discrepancies which did occur seemed to be accounted for by the supposition that his general condition since he had been at Netley had greatly improved. His story hung together so well, and he really appeared so frank, and so anxious to get well, that a few days after admission he was transferred to the convalescent ward, and given parole.

On 23rd October 1916 he broke his parole, and twenty-five days later was arrested by the civil police and returned to the hospital. The police reported that he had been masquerading in the borough and district as a wounded French soldier who had been attached as interpreter to the British Army in France. He had imposed himself upon quite a number of people with his tale, and had in his possession two lines of leaden type as follows: "Interpreter R. le Auldere, attached to the 1st Division."

Acting on the above information, it was thought advisable to again obtain from the patient a rather more comprehensive review of his life.

As formerly, he stated that he had been born in France, that his father and mother were now both dead, that his sister had died of consumption, that one brother, an explorer, had contracted and died of "black-fever" while in Africa, and that another brother, a petty officer in the mercantile marine, had been drowned at sea. He himself had been a strong, healthy, intelligent boy who had got on well at school, and at the age of sixteen years

passed an examination in "mathematics, writing and dictation, geography, grammar, geometry, metric system, decimals, and drawing," so as to enable him to enter a military academy at Paris. While at the military academy he was instructed in the planning of forts and dockyards, in the mechanism of guns, in sketching and drawing, and this he rather naively remarked was "so as to point out harbours in case of being called on to become a spy." He then told about not liking the restrictions of the military academy, about quarrelling with his father on account of his drunken habits, and of running away to sea.

He now, however, stated that on arriving in England at the age of 16 years he deserted his ship, made the acquaintance, at Pembroke Dock, of a French lady who knew his mother, and became an adopted son. Up until 1908 he stayed with this family, but, in the meantime, employed himself as a farm-hand with a gentleman in Honeyborough, and after two years with him received employment with Sir Owen Phillips at Lorennny Castle, for whom he worked up until 1908. On account of his drunken habits he had a quarrel, went to Bristol, and in 1908 enlisted in the 1st Gloucesters. Shortly afterwards he went to Malta with his regiment, where he was for a period of three years, returning to England in 1911. He was for a time at Portsmouth, then was transferred to Bristol, and was on duty there when the war broke out.

He now denied ever having said that he had been married and divorced from a Frenchwoman, but said that while in Bristol he had married an English girl, who had divorced him on the grounds of drunkenness and cruelty, but that she was now dead.

He again stated that he had gone to France with his regiment in August 1914, but in January 1915 had been invalided home on account of "trench feet," and eventually was discharged from the army as physically unfit. In June 1915 he re-enlisted in the Durham Light Infantry, and in January 1916 was again ordered to France. He remained well until July 1916, when he was blown up on the Somme by a shell explosion, and following this could remember nothing until he found himself in No. 3 General Hospital. He remembers being accused of desertion, but sentence was not passed, as he was held by the medical officer to have been irresponsible. (As a matter of fact he was, at that time, considered to be a case of dementia præcox.)

In regard to his twenty-five days' absence from this hospital, he stated that while in the town he had met one or two of his old regimental comrades, that he had got drunk, and that his friends had taken him to Manchester with them. It was while he was attempting to get back to hospital, "as he was longing to return to duty," that he was arrested by the civil police.

This story was so manifestly at variance with the ones already told that the patient was accused of being untruthful and of wilfully lying. He at first stoutly denied that such was the case, maintained that he had told the previous stories because he did not feel that it was necessary for him to give the details of his life to the medical officer, and asserted that the last story was a true and faithful account.

Even when confronted with a criminal record, which had been obtained from the police, he continued to maintain his innocence, and in reference to a period of confinement in a Borstal Institution, he pointed out that such was impossible owing to his being over age for such a place at the time the offence with which he was charged was committed. He alleged that his record had been mixed up with the record of an adopted brother, who was a criminal. Later, however, he was prevailed on to give the following *approximately* true story of his life, which is in striking contrast to the preceding.

He was born at Weyland, in Pembrokeshire, in 1890, and is now 26 years old. His father was a shipwright, and he himself was the fifth youngest of a family of thirteen. He was always strong and healthy, had a comfortable home, but was always in difficulty with his parents owing to his refusing to go to Sunday school, Band of Hope meetings, &c. He describes himself as always having been of a roving disposition, his family could never keep control of him, and even at the early age of 7 years he had run away from home and gone to relatives at Haverford West. At school he got on well, was a favourite both with comrades and teachers, and learned easily. He was a voracious reader, and was principally interested in books of adventure. He had always had a great liking for the sea, but his parents would not allow him to go, owing to one of his brothers having been drowned at sea. He admitted having early acquired a taste for drink, but in regard to sexual habits he made the rather paradoxical statement, "That is one thing I am innocent of doing, simply

because I believe a woman to be a higher thing than what she is."

When at school he reached standard VII., and after leaving took up work as a gardener. When he was about 16 years old he ran away to Pontypool but was eventually returned home. One month later he again left home, was convicted of drunkenness at Haverford West, and was ordered to pay a fine of 7s. 6d.; this, he asserts, was his first offence. Following this he went "straight" until 1910, when he was arrested at Pembroke Dock for stealing milk, and was sentenced to a reformatory for three years. After about three months he and a comrade organised a mutiny and escaped. In January 1911, while more or less on his beam-ends, he stole some money, and was sentenced to three months' imprisonment, after which he was handed over to the reformatory authorities. In 1913 he was released from the Reformatory, and enlisted in the army in the 1st Gloucesters. In a few months he deserted, and in January 1914 he broke into a warehouse and stole some clothing, but was arrested and sent to a Borstal Institution for three years. While there he got to know that he would be allowed out if he offered to rejoin the army, so he wrote to his former regiment, "offered," as he says, "his services to King and Country," and eventually in June 1915 he reported in London and was drafted to the Durham Light Infantry. Owing to some error about him he was, after two months, arrested as a deserter from the Gloucesters, was kept in the guardroom for forty-eight days, and then the case was dismissed. Owing to the fact that he felt that he had been unjustly treated he stole the uniform of a 2nd lieutenant, deserted, but was arrested by the Darlington police, and sentenced to six months' imprisonment. In January 1916 he was released from prison, was sent back to his battalion, and two days later was put on a draft and sent to France. He served there until August 1916 when he was "shell-shocked," was sent to a Field Ambulance, No. 3 General Hospital, Netley, and Lord Derby War Hospital. He remembers being court-martialled for desertion, but nothing came of it on account of the medical evidence.

After breaking his parole from this hospital he masqueraded in the district not only as "R. le Auldere," but also as "Le Marchal," and undoubtedly imposed on various people. For instance, one day at the railway station he carried his right arm

in a flexed position underneath his coat, and told a school teacher, who took compassion on his seeming loneliness, that he was a French soldier who had been wounded in the right arm at Verdun, and at present was out on leave from a convalescent hospital in the neighbourhood. On the strength of his story this kind-hearted lady invited him to tea at her house, and showed him other kindnesses. To others he stated that he had been a teacher of languages at Cambridge University, that he had a son being educated there, that he had formerly been a teacher at Marseilles, and, of course, that he was attached to the British Army as interpreter. The police report stated: "As a liar this man is somewhat of a phenomenon, but to the ordinary man he shows no sign of mental weakness, and exhibits himself as he is—a thorough rogue, an accomplished liar, an impostor, and, when cornered, a foul-mouthed loafer."

It is unnecessary for me to mention any other details except to say that in contrast to, and no doubt as compensation for, his moral defects, he shows an easy superficial piety which is expressed not only in his conversation, but also in his letters. For instance, shortly after his return to this hospital, he requested an interview, and stated how he was much concerned about a certain young lady of his acquaintance who had gone wrong, and asked for leave so that he might go out and marry her, and thus save the honour of her family. In a letter to her he finishes by saying: "I therefore commit you to the care of our dear Lord and Saviour Jesus Christ, your ever-loving sweetheart." The contrast, too, is well shown in the following letter:—

"MY DEAR FRIEND,—Just a few lines, hoping to find you in the best of health. I am sorry I did not write to you before, but I have not had time until I came into hospital. I have had a rough time of it since I went to the front. I should like to hear from some of my old Sunday school chums, as I may be able to see how many are enlisted, and are doing their duty, as I am very interested with L, for I very often want to know how the people are getting on there. Have you built the new chapel that you said was going to be built? I hope to be sent back to my regiment before very long, and then I will come and visit you and the old school and chapel. Your sincere pupil."

From the point of view of diagnosis the features which seem to stamp such a case as this as one of high-grade mental deficiency

are:—(1) Precociousness, as seen by the facility with which ordinary school knowledge was acquired, and the interest in books of adventure; (2) the roving disposition, with inability apparently to concentrate; (3) the blunting of the emotional tone, as evidenced by the absence of any real consciousness of guilt or shame, and the lack of any particular affection towards his family; (4) the lying, but in contrast to it the absence of adequate precautions to prevent the lies from being detected; (5) the rather attractive personality, and (6) the total irresponsibility of the individual.

The fact that the memory of these individuals for their acts of wrong-doing is intact, the absence of anæsthesia, and of any splitting of consciousness readily differentiates such conditions from hysteria and epilepsy.

A harder question to settle, however, is that brought up by the police report in which it is stated that to the ordinary man he shows no sign of mental weakness. How is it possible then to distinguish such a case from one of wilful criminality? I may be wrong, but to my mind the true criminal plans his work with a thorough knowledge of the risks he is taking, and fully realises the punishment in store for him should he fail or be arrested. In other words he formulates beforehand, and weighs in the balance the risks which he is going to take, with the possible gain which may accrue to him, and then acts towards the given situation in the same way as a normal adult man would do when called upon to meet some more or less unexpected difficulty.

Cases of high-grade mental deficiency, on the other hand, although they may be adults in years, retain their childishness in that they sacrifice their future for the gratification of their immediate needs, and thus act with utter lack of foresight and total irresponsibility. Dr Jane Robertson, in a recent most excellent paper on this subject, has put the matter in this way: "It is only after prolonged experience of the insane irresponsibility of these cases that they are sometimes considered in a proper light, and it is fortunate indeed if by that time the patient has not found himself in prison, or suffered social ostracism, acknowledged of no man, and accepted of no institution. He has all the mannerisms of sanity, but none of its principles, and unfortunately his mannerisms are accepted at their face value by most people until betrayed by the lack of principle, *i.e.*, the insanity; and then it is the

machinery of punishment, of retribution, not of sympathetic analysis, that is promptly set in action against him."

At a time such as this, when the feelings of the public have been strongly roused to do everything that is possible for the welfare of our wounded soldiers, the attractive personality, the easy address, and the plausible lying of such a case as the one presented constitutes a very real danger, and it behoves us, therefore, to attempt to safeguard the public by adopting some method for repression. But what is it possible to do? It would be manifestly unfair both to the public and the patient to return such a case to the army, owing to the fact that it is more or less certain that he would prove to be quite inefficient as a soldier, would no doubt in a short period of time not only be a trouble and danger to himself, but also would be a danger to others, and a burden on the State. We have no charge against him whereby it would be possible to hand him over to the police, and so far as his mental condition is concerned it would be impossible, with our present laws, to commit such a patient to an asylum.

In addition it has been over and over again emphasised that prison life, instead of acting as a deterrent, tends to act as a sort of hardening process, and practically means nothing, as such cases are practically impervious to any form of punishment. It is true that in many asylums one, from time to time, comes across such patients, who have been committed principally on account of their past record, and maybe a transitory outburst of irritability, but one does not need to have had much experience of asylum work before it is clearly brought home that such patients are a focus of infection to all around them, and exert a most malign influence.

There seems to be no doubt that what we must strive to do is to get such cases before they have started on their criminal course, and to that end parents, school teachers, and those who administer justice in our courts for juvenile offenders should have some knowledge of the problems presented by such cases. In this connection it might be mentioned that, attached to the juvenile court at Chicago, an expert psycho-pathologist in the person of William Healy has done most excellent and most interesting work.

Such cases are capable of doing such splendid work that, not only from the philanthropic standpoint but also economically, it will repay the State to segregate such individuals in colonies,

where they would be educated and trained to become useful citizens.

I wish to thank Lieutenant-Colonel Simpson, R.A.M.C., for permission to publish this case.

Abstracts

BRAIN, SKULL, &c.

THE DISPOSAL OF CASES WITH HEAD WOUNDS. (*La conduite (169) à tenir vis-à-vis des blessures du crâne.*) M. PIERRE MARIE, *Rev. Neurol.*, 1916, xxiii., April-May, p. 453.

THIS is the account of a meeting of the Paris Society of Neurology at which representatives were present from military neurological centres of France and allied countries. The discussion was opened by M. Marie, who limited the field to the more obscure subjective phenomena present in cases with head wounds. These comprised abnormal sensations, often limited to the site of the lesion, and headache. In addition, transitory cloudings of consciousness may occur, and dizziness, which is distinguished from true vertigo by the absence of change in the relation of patient to environment. Besides these there are changes of character, insomnia, incapacity for work, memory troubles, and vaso-motor symptoms.

M. Maurice Villaret drew attention to those cases which had apparently recovered. He maintained that careful examination revealed in almost all these patients residual symptoms. In occipital lesions, visual troubles; in parietal cases, unilateral astereognosis; and in any lesion, epileptic equivalents and mental disturbances may be present. He concluded that only cases without these residua can be fit for field service, under medical supervision. X-ray examinations should be carried out carefully in every case.

M. Henri Claude confirmed the frequency of subjective phenomena from a study of over 400 cases with head wounds. He laid stress on the bad effects the noise of explosions had on these patients. Increase of albumin in or hypertension of the cerebro-spinal fluid may be found where localising signs are absent. He was of opinion that very few cases can be sent back to the firing line, and these never to positions requiring initiative and sang-froid.

M. J. A. Sichard remarked that he had found pathological changes in the cerebro-spinal fluid in more than a quarter of a hundred cases examined. He considered such cases unfit for military service.

M. P. Collier had made a search for some objective sign present in cases complaining of subjective disturbance, and claimed to have found unilateral or bilateral mydriasis in two-thirds of 79 cases. He considered this sign of importance in determining the reality of the subjective trouble, and ascribed the mydriasis to meningeal irritability.

M. J. Babinski stated that he had often found labyrinth vertigo in cases with head wounds. In such cases the question of malingering can be dismissed, and they can be relieved by repeated lumbar puncture. He gave it as his opinion that no cases with head wounds should be returned to field service.

M. J. Froment, in conjunction with M. Babinski, had studied cases with fracture of the left parietal bone. They discovered that after hemiplegia and aphasia had cleared up, certain intellectual functions remained impaired, and such cases were considered unfit for active service.

M. Castan had examined trephined cases with a view to determining symptoms common to all, and these symptoms are almost identical with the subjective troubles mentioned by M. Marie. Of 106 cases examined, 34 recovered, and 3 were sent back to the front. The equilibration disturbances of these last 3, however, became much worse under the explosions. He suggested that men who recover should be attached to sanitary units, where the medical officer can both apportion their duties and supervise them.

M. André Léri reported that he had found subjective symptoms mostly in cases with frontal or occipital lesions. The headache is usually frontal, even when the lesion itself is occipital. He considered meningeal adhesions to be at the bottom of them, especially where adhesions were between membranes and brain. He had, therefore, initiated treatment by ionisation, using potassium iodide, but data to judge results were not yet available. These adhesions were very tenacious, and their duration might extend to a year. He had come to the conclusions: (i.) that malingering should not be assumed in these cases; (ii.) that they should not take up hospital beds; and (iii.) that auxiliary service might be tried for the lighter cases, but that it might aggravate more serious ones.

M. Georges Guillain had had under observation patients with these subjective symptoms in the trenches. He was of opinion that no officer afflicted in this way should be sent back to the

firing line, since on him so much depended, whereas wider liberty of judgment was permissible with men.

M. Marcel Briant remarked that tachycardia, often increased by touching the scar, was sometimes present with the subjective symptoms, and M. Pitres added that the sympathetic system could be involved.

The general consensus of opinion seemed to be that cases with head wounds, and having only subjective symptoms, were fit only for auxiliary service. If a patient seemed to have recovered, it was justifiable to send him back to the firing line only after the most careful examination for residual troubles had proved negative.

H. W. HILLS.

EARLY OPERATION IN WOUNDS OF THE HEAD. (*L'opération primitive des blessés du crane.*) *Rev. Neurol.*, 1916, xxiii., June, p. 721.

At this combined meeting of the National Society of Surgery and the Neurological Society of Paris, six main topics are fully discussed. It is impossible to adequately abstract these most interesting discussions, but some of the main points may be mentioned, and the scope of the paper indicated.

In regard to the question of early operation in head cases, the general view is held that all cases, whether they are simply scalp wounds or involving the deeper structures, should be operated on at once. The various types of operations are discussed. Other points which are discussed under this heading are: Where there is a simple fissure of the outer table must splintering of the inner table always be allowed and sought for? Is the flap incision preferable to the crucial? What are the results, the indications, and the technique for the immediate removal of intra-cerebral foreign bodies?

The second main question is in regard to the removal of cases of head injury. The consensus of opinion was largely in favour of having them taken as rapidly as possible to well-equipped hospitals in the rear where quiet could be obtained, and where everything was in readiness for dealing with emergencies.

The third question concerns itself with hernia and abscess of the brain the meningitides, and epilepsy; the frequency, prognosis, and treatment of these conditions are dealt with.

The fourth question is devoted to a consideration of the development of the more remote phenomena (motor, visual, and subjective disorders) to which cases of head injury are exposed and the chances of recovery and the duration of the illness are discussed.

The fifth and sixth questions have to do with the operation of cranioplasty, and the protective value of the steel helmet.

D. K. HENDERSON.

**DISTURBANCES OF VISION FROM CEREBRAL LESIONS, WITH
(171) SPECIAL REFERENCE TO THE CORTICAL REPRESENTA-
TION OF THE MACULA.** GORDON HOLMES and W. T. LISTER,
Brain, 1916, xxxix., p. 34.

DETAILS are given of twenty-two cases in which wounds of the occipital region caused defects in the visual fields. The cases are divided into four groups, which deal with quadrantic defects, central scotomata, homonymous hemiopia with central scotomata, and paracentral scotomata respectively. In another case a wound traversing both lower parietal areas produced extreme contraction of the fields, with retention of a small central area of less than 10°.

The cortical centre for macular vision is tentatively placed in that portion of the area striata which extends to the margin and on to the lateral surface of the occipital lobe. The macular area is not represented bilaterally in the cortex. It is of interest to note that the author's work supports the view that achromatopsia does not occur with intact vision for white, in so far as they found that their observations provided no conclusive evidence that achromatopsia, with vision for white unimpaired, is produced by cerebral lesions which involve either the cortex or the optic radiations. The author's conclusions, which are not to be regarded as final, are as follows:—

(1) The upper half of each retina is represented in the dorsal and the lower in the ventral part of each visual area.

(2) The centre for macular or central vision lies in the posterior extremities of the visual areas, probably on the margins and the lateral surfaces of the occipital poles.

(3) That portion of each upper portion of the retina in the immediate neighbourhood of, and including the adjacent part of, the fovea centralis is represented in the upper part of the visual area in the hemisphere of the same side, and vice versa.

(4) The centre for vision subserved by the periphery of the retina is probably situated in the anterior end of the visual area, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forwards in the visual area.

The paper is well illustrated, full of interest, and marks a distinct addition to the accuracy of our knowledge of cerebral localisation. It should be read in the original by all who are interested in the subject.

H. M. TRAQUAIR.

A CASE OF OBLIQUE HEMIANOPIA FROM WOUND OF OPTIC (172) CHIASMA. PURVES STEWART and A. D. GRIFFITH, *Lancet*, 1916, ii., July 15, p. 104.

A SHRAPNEL fragment had destroyed the right eye, and was found by radiography to be lying "about a quarter of an inch above and a quarter of an inch in front of the upper limit of the sella turcica." The patient was unconscious at first, and nearly died of septic meningitis. Complete blindness was present in the left eye, the optic disc being normal. After nearly four weeks the remains of the right eye were removed, and a month after the injury sight began to return on the nasal side of the field of the left eye. Two months after the injury the optic disc was atrophic, and the pupil widely dilated with a typical hemiopic reaction. The field of vision showed an irregular hemianopia, the dividing line passing 30° to the inside of the vertical meridian below, and nearly as much to the outer side above. The periphery of the upper nasal quadrant was also lost, and a small tongue of retained vision projected into the blind temporal field just above the horizontal meridian. Eleven weeks after the injury the pupil contracted when light was thrown on the nasal retina; also, although not so briskly, as on stimulation of the temporal side.

After glancing at the course of the optic fibres from retina to tract the authors point out that a lesion of the right tract would not have produced the field conditions found. In a diagram the site of the lesion is indicated at the left side of the anterior margin of the chiasma, just at the termination of the left optic nerve. It is not clear whether the diagnosis of chiasmal lesion was founded on the radiographic evidence or on the field of vision. If by "upper limit of the sella turcica" is meant the tip of the dorsum sellæ, the position stated would be approximately that of the anterior margin of the chiasma, otherwise the position would be farther forward in the region of the optic nerve.

The field conditions depicted might equally well have been due to the results of a lesion of the inner side of the optic nerve in front of the chiasma, which is also suggested by the history of complete blindness for nearly a month. The diagnosis from visual symptoms of a traumatic lesion of the chiasma in a one-eyed subject must be a matter of extreme difficulty, and it is to be regretted that the authors have not gone more closely into their reasons for selecting the point indicated.

H. M. TRAQUAIR.

LATE APOPLEXY FOLLOWING SHELL EXPLOSION WITHOUT**(173) EXTERNAL WOUND.** (*Apoplexie tardive consécutive à une commotion par éclatement d'obus sans plaie extérieure.*)G. GUILLAIN and J. A. BARRÉ, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1473.

A SOLDIER lost consciousness for several hours as the result of an explosion of a large shell close at hand, and remained in an asthenic condition, though gradual improvement took place. The cerebro-spinal fluid was normal. A month after the explosion he suddenly developed subintractant epileptiform attacks, and became comatose. Death took place the same day. The autopsy showed diffuse congestion of the brain, most marked in the right hemisphere, in which a recent hæmorrhagic focus was found situated in the anterior part of the lenticular nucleus and in the front limb of the internal capsule.

The pathogeny of this late apoplexy is as follows:—

The explosion had primarily caused a slight hæmorrhage from a vessel in the central grey nuclei in the right hemisphere, this hæmorrhage explaining the loss of consciousness and subsequent symptoms. The vascular lesion had not completely healed, and a month later a secondary hæmorrhage had occurred causing the ictus epileptiform attacks and coma.

J. D. ROLLESTON.

TWO CASES OF ORGANIC HEMIPLEGIA FOLLOWING THE**(174) BURSTING OF HEAVY EXPLOSIVES WITHOUT EXTERNAL WOUND.** (*Deux cas d'hémiplégie organique consécutive à la déflagration de fortes charges d'explosifs sans plaie extérieure.*)G. GUILLAIN and J. A. BARRÉ, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1470.

In the first case there was a severe left hemiplegia with disturbance of superficial and deep sensibility and early contractures. The diagnosis on admission to hospital was "hystero-traumatism, hysterical left hemiparesis." In the second case the hemiplegia was slight, and would doubtless clear up without leaving any trace. In both cases the changes in the cerebro-spinal fluid were slight; in the first patient there was hypertension with slight lymphocytosis and without excess of albumin, and in the second there was a moderate excess of albumin without obvious hypertension and without lymphocytosis.

The pathogeny of these organic hemiplegias is explained by the existence of small hæmorrhagic foci in the course of the pyramidal tracts.

J. D. ROLLESTON.

HEMIPLEGIA FOLLOWING INTOXICATION BY ASPHYXIATING

(175) **GASES.** (*Hémiplégie consécutive à une intoxication par les gaz asphyxiants.*) L. GIROUX, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1486.

A HITHERTO healthy soldier, aged 20, was the victim of a gas attack while asleep. In twenty-four hours he lost consciousness and remained for a week in a comatose state, on recovery from which he was found to have a flaccid left hemiplegia, in which contraction subsequently ensued. Giroux attributes the hemiplegia to the thrombotic action of the chlorine gas. J. D. ROLLESTON.

FOUR CASES OF HEMIPLEGIA CAUSED BY EMBOLISM

(176) **FOLLOWING GUNSHOT WOUNDS OF THE CAROTID ARTERIES.** L. COLLEDGE and J. SHAW DUNN, *Lancet*, 1917, i., Jan. 13, p. 57. (2 figs.)

IN each of these cases, all of which proved fatal, the hemiplegia and cerebral disturbance were caused by occlusion of the middle cerebral artery or its branches. In all four there was thrombus present in the carotid arteries in the neck, consequent on damage of the arterial walls, and in each there were several inches of the internal carotid artery patent between that thrombus and the portion blocking the cerebral vessel. The occlusion of the cerebral vessels consequently occurred by embolism, and similar emboli were found in one instance in the branches of the external carotid also. The damage to the large cervical arteries was in every case considerable. In three there was actual perforation of the wall; in the fourth the trunk was found completely severed, but this, no doubt, occurred secondarily by sloughing after the vessel was fully thrombosed. In only one case was ligature necessary.

Makins (*v. Review*, 1916, xiv., p. 449) drew attention to the tardy development and partial nature of the paresis as outstanding features in his series, and contrasts this with the prompt and complete hemiplegia, followed by a fatal issue in twenty-four to thirty-six hours, which may follow after ligature of the common carotid artery. In these four cases the early appearance and completeness of the hemiplegia and the rapidly fatal issue resemble the effects of ligature of the common carotid. A. NINIAN BRUCE.

STUDIES ON LABYRINTHINE DISORDERS IN CONCUSSION

(177) **AND WOUNDS OF THE HEAD.** (*Recherches sur les troubles labyrinthiques chez les commotionnés et blessés du crâne.*) FRANCOIS MONTIER, *Rev. Neurol.*, 1916, xxiii., July, p. 9.

SEVERE vertigo, either accompanied or unaccompanied by disorders of hearing or gait, is quite common in cases of concussion or of head injury, but the non-recognition of this fact not infrequently

permits such cases of being unjustly accused of simulation or exaggeration. It is principally on this account and from this point of view that eighteen cases of concussion and twenty-eight cases of wounds of the head have been studied. In the twenty-eight cases where the head was injured it was the temporo-parietal region which was most frequently affected. 87 per cent. of those forty-six cases showed labyrinthine symptoms.

The tests principally used were the voltaic or Babinski's test and the caloric or Barany's test.

The technique of these tests is fully described, and the results obtained are given in detail. In general it may be said that practically identical results were obtained with each test, but the author feels that the voltaic test is the more precise and the easier to apply and read. In contrast to the findings in the temporo-parietal cases in three cases of traumatism to the occiput it was found that there was lowering and not increase of resistance to the voltaic current, that the head inclined either directly forward or backward, and not laterally, that the caloric test was as in normal cases, and that there was inversion of the "pointing" test. These findings seem to the author to differentiate conclusively between cerebellar and labyrinthine disorders.

D. K. HENDERSON.

MOTOR APHASIA IN FORENSIC MEDICINE. (*Afasici motori in* (178) *medicina forense.*) G. D'ABUNDO, *Riv. ital. di Neuropatol., Psichiatr. ed Elettrotet.*, 1916, ix., p. 463.

A CARABINEER, aged 40, who had contracted syphilis sixteen years previously, received a gunshot wound in the left frontal region in May 1914. When examined by D'Abundo in September, he presented right hemiplegia with contracture and muscular hypotrophy amounting to muscular atrophy in the muscles of the right hand, motor aphasia and dysgraphia, and signs of syphilis in the mucosæ and glands. Some improvement in the aphasia took place on re-education.

At the Assize Court, held two years after the attack, the patient recognised two of his assailants, who were consequently sentenced to long years of imprisonment. This case is of considerable psychiatric interest, inasmuch as it shows that the mental deficit generally attributed to motor aphasia by P. Marie is not acceptable.

D'Abundo maintains that a decision as to the mental state in motor aphasia must be made in each individual case, because the age of the patient, the pathological conditions of the arteries, the extent of the lesions, and the association of special morbid conditions in other organs may be sufficient to cause a real mental deficit.

J. D. ROLLESTON.

SPINAL CORD, &c.**THE TREATMENT OF GUNSHOT WOUNDS OF THE SPINE.**

(179) ALFRED J. HULL, *Journ. Roy. Army Med. Corps*, 1917, xxviii., Jan., pp. 66-77. (4 plates.)

THE majority of cases of gunshot injury to the spine require excision of the wound and exploration at the earliest opportunity. The diagnosis is not so gloomy as past experience teaches. Local anæsthesia is practically essential. The administration of urotropine should be begun as soon after the injury as possible. The presence of a missile, together with severe pain, are indications for immediate operation. Accurate localization of foreign bodies is of the utmost moment. A lateral as well as an antero-posterior view is desirable.

A. NINIAN BRUCE.

THE SYNDROME OF CRANIO-SPINAL HYPERTENSION
 (180) **FOLLOWING CONTUSIONS OF THE CERVICAL VERTEBRÆ.**

(Le syndrome d'hypertension céphalo-rachidienne consécutif aux contusions de la région cervicale de la colonne vertébrale.)

HENRI CLAUDE and H. MEURIOT, *Le Progrès Médical*, 1916, Dec. 5, No. 23, p. 223.

THE writers draw attention to a syndrome occurring in subjects who have received a violent traumatism of the posterior cervical region which has caused contusion or more or less concussion of the cervical spinal cord. Sometimes there is also present a partial fracture of a vertebra; after a rather long period the following syndrome appears:—(1) General signs of intracranial hypertension, viz., headache, vomiting, vertigo, venous hyperæmia of retina or papillary stasis, and signs of labyrinthine irritability, &c.; (2) localized signs of nervous or radicular compression, due to increased pressure of the cerebro-spinal fluid in the cervical region, viz., signs of sympathetic irritation (oculo-sympathetic syndrome, exophthalmos, mydriasis), sensory disturbances in certain cervical root areas, and specially in the first thoracic and the lumbo-sacral root areas, with consecutive changes in the tendon jerks. Lumbar puncture quickly leads to recovery, but it may have to be done more than once. Three cases of this syndrome are detailed: in the first case there was with the oculo-sympathetic syndrome a paresis of the homo-lateral inferior oblique muscle of the eyeball; in the third, signs of labyrinthine hyper-excitability with inequality of pupils. The writers think that too little attention has been paid to spinal hypertension. They recommend that in every case of cranio-vertebral traumatism one should

examine the ocular fundi and the labyrinths; and they point out that, unless the importance of spinal hypertension be realised, one would be tempted to attribute the whole symptomatology to a medullary or radicular lesion for which one would not think of surgical operation.

LEONARD J. KIDD.

GENERAL CONSIDERATIONS ON CAMPTOCORMIA. (*Considérations générales sur la camptocormie.*) MAIE ROSANOFF-SALOFF, *Nouv. Icon. de la Salpêtrière*, 1916-17, No. 1, p. 28.

THE author describes a series of cases of acute flexion of the trunk in an anterior direction, occurring in soldiers as a result of being blown up by a shell, or of being buried, with a loss of consciousness of variable duration. The first symptom is lumbar pain, aggravated by movement: consequently the patient immobilises his back, and finds the position of greatest relative comfort in bending forward till the head is almost between the knees. This develops a fixed attitude in neuropathic subjects, as all such patients are. The prognosis is good, provided the cases are treated early in a suitable hospital with proper military-medical regime. The patient is put in a plaster jacket, with or without an anæsthetic, which is kept in position for eight, ten, or fifteen days. This treatment, coupled with proper discipline and an atmosphere that of itself suggests cure, succeeds without fail in the writer's experience. Camptocormia is derived from κάμπτω, to bend, and κορμός, the trunk.

S. A. K. WILSON.

THE BLADDER IN GUNSHOT AND OTHER INJURIES OF THE SPINAL CORD. (Hunterian Lecture.) J. W. THOMSON WALKER, *Lancet*, 1917, cxcii., Feb. 3, p. 173. (3 figs.)

THIS paper is based upon a study of 111 cases treated in the Star and Garter Hospital (65 beds), and 339 cases at the King George Hospital.

After a short account of the anatomy and physiology of micturition, four bladder states are described:—(1) *Retention of urine* from paralysis of the detrusor muscle, leading to (2) *retention with overflow* (passive incontinence) where only the surplus beyond a certain large accumulation escapes. (3) *Periodic reflex micturition* (active incontinence), where a reflex discharge of urine takes place whenever a certain quantity of urine accumulates. The condition is that of the infant's bladder, cerebral control being absent. It is also found after section of the cord above the lumbar micturition centre. (4) *Paralytic incontinence*, where the bladder is flaccid

and the sphincter atonic; the urine dribbles away from the bladder without accumulating. This is said to occur when the lumbar centres are destroyed.

Complete retention was found to occur, at the commencement, in all cases where micturition was affected, at whatever level the injury might be, and it occurs in cauda equina lesions as well as in cord lesions. Periodic reflex micturition is the second phase in all lesions of the cord, not excepting those of the lumbar enlargement. Periodic reflex micturition develops in more than half the cases of cauda equina lesion, but occasionally voluntary micturition follows directly on a period of complete retention.

The most common and most fatal complication in a paralysed bladder is infection. Over 90 per cent. of cases of spinal injury at the Star and Garter Hospital have a serious infection of the urinary tract, and it is the usual cause of death. This infection usually takes place within the first week after the injury. A hæmorrhagic cystitis usually results, which ascends to the renal pelvis, causing pyelitis and septic pyelonephritis.

Treatment of the urinary tract in paraplegia resolves itself into two chief lines: (1) Provision for the removal of urine and (2) treatment of septic complications. In the early stages, with complete retention, three methods are available: (i.) Catheter life, (ii.) tied-in catheter (catheter "à demeure"), and (iii.) suprapubic drainage. The septic complication may be treated by (i.) local treatment by bladder washing, (ii.) medicinal treatment, and (iii.) vaccines.

The ascent of infection from the bladder to the kidney is due to pressure or intravesical tension, aided by diminished resistance to bacterial invasion on account of the nerve lesion. It must be remembered that motile bacilli may travel upwards even against the stream. Over-zealous washing of an inflamed bladder with too strong antiseptics, or too great distension of the bladder, will just as readily produce an ascending infection as a septic catheterisation of the original over-distended bladder.

The remedy lies in early and continuous drainage of the bladder. Continuous urethral drainage by catheter is unsuitable, as it quickly sets up a urethritis. A prophylactic suprapubic cystotomy should be performed at the earliest possible moment, before any catheter has been introduced, and the bladder drained continuously until the second stage of active incontinence is reached. The tendency to an ascending infection from urinary tension is thus avoided. Whether this is practicable has yet to be determined.

Suprapubic cystotomy to drain a septic paralysed bladder, or when the urethra is œdematous and bleeding from catheterisation,

will quickly clear up cystitis if no source of recurrent infection such as pyelonephritis, which tends to constantly reinfect the bladder, is present. If the cystitis is well marked and recurrent attacks of pyelonephritis occur, drainage of the bladder by abolishing intravesical tension should prevent the recurrence of the ascending infection.

A. NINIAN BRUCE.

NERVES.

THE VALUE OF CLINICAL SIGNS IN RECOGNISING WOUNDS
(183) **OF THE PERIPHERAL NERVES.** (*La valeur des signes cliniques permettent de reconnaître dans les blessures des nerfs périphériques.*) *Rev. Neurol.*, 1916, xxiii., April-May, p. 477.

THE complete section of a mixed nerve is followed by immediate and total loss of voluntary movement, by loss of tonus, loss of reflexes, and then later atrophy of the muscles supplied by the severed nerve. No irritative phenomena are present, and if there should be spasm, tremor, or convulsive movements, an incomplete sectioning of the nerve or an irritant would be indicated. The fibro-tendinous and articular rigidity which may sometimes be seen is due to prolonged immobility in bad positions. Emphasis is laid on the help which may be obtained by mechanical tapping of the muscle mass; when one fails to get any response, one may be sure that the muscle is irrevocably lost.

In regard to the sensory disorders, stress is laid on the absence of all pain on pressure of the nerve trunk below the point of section. In complete section pain is practically always absent, and such cases never suffer from the horrible pain met with in cases of incomplete section. Painful paræsthesias are also practically never met with in complete section. The familiar types of vaso-motor and trophic disturbances are also mentioned.

In the second part of this paper the clinical signs of restoration of function are discussed. The first clinical signs of a return of function are the reappearance of muscular tonus and involuntary contractions, then voluntary movement, sensation, and the tendon and cutaneous reflexes. Every nerve, of course, has its own specific functions, and these should always be carefully investigated in deciding whether a cure has or has not taken place.

The discussion following on the paper is reported in full.

D. K. HENDERSON.

GUNSHOT WOUNDS OF PERIPHERAL NERVES. BYRON STOOKEY,
(184) *Surgery, Gynaecology, and Obstetrics*, 1916, xxiii., Dec., p. 639.

THIS paper is based on a study of seventy-five cases of nerve injuries and has thirty-four figures. Conclusions:—

1. With the use of high explosives and bullets with high velocity the frequency of nerve lesions has increased.

2. Peripheral nerves may be injured by direct violence of projectile, and by the violence imparted to bits of bone or even foreign bodies.

3. Peripheral nerves may be implicated secondarily by scar tissue, or callus, or both.

4. Diagnosis cannot be made before operation between anatomical and physiological division. Diagnosis can usually be made in cases with incomplete division.

5. In war surgery primary suture is rarely possible due to infection.

6. Exploratory operation is indicated when a diagnosis of complete division is made. Delay in operating usually means delay in return of function.

7. Nerve-freeing is in many cases to be preferred to excision and suture. When the nerve is widely implicated and there is a large loss of continuity, it is better to do nerve transference or nerve transplantation than tubulisation or suture with the nerve under tension.

8. Stretching of the nerve should not be done, as it causes karyolysis of the nerve cells in the ventral horn with subsequent degeneration of the nerve axon in the proximal nerve trunk.

9. Efficient splinting to prevent contractures and overstretching of muscles is imperative, both before and after operation.

10. The terms epicritic and protopathic tend to be confusing. Greater accuracy in use of specific terms, as area of cotton-wool, area of pin-prick, areas of moderate and extreme degrees.

11. Musculo-spiral nerve injured in its lower third does show loss of sensation on narrow band over dorsum of thumb, usually only loss to cotton-wool and temperature sense.

12. Injury to musculo-spiral nerve may cause dissociation of temperature sense in the area on dorsum of hand,—without loss to cotton-wool.

13. The median nerve does not supply any skin on the dorsum of the thumb; supplies up to line in continuation of lateral borders of nail.

14. The anterior cutaneous division of the ulnar nerve supplies the skin in the same manner as does the median nerve, *i.e.*, on to dorsum of fifth and part of fourth, middle and distal phalanges. The posterior cutaneous division supplies the ulnar side of the hand, and the proximal phalanx of the fifth and part of the fourth.

15. The action of extensors assists the interossei in separating

the fingers. To test for paralysis of the interossei have the patient bend his fingers at right angles at metacarpal phalangeal joints; prevent effort of extensors by holding the fingers across; then have patient try to separate his fingers gently.

16. Return of motor function begins with the muscles which first receive their supply below the lesion. The return is earlier the nearer the lesion is to the periphery.

17. Trophic ulcers occur only after trauma. Their repair appears to be no different from that in other parts.

18. Functional disorders may be superimposed on organic peripheral nerve lesions. Usually they are readily recognised. (Nerve injuries are far more frequent in the upper than in the lower extremities.)

LEONARD J. KIDD.

WAR INJURIES TO THE MUSCULO-SPIRAL NERVE. W. B. (185) WARRINGTON and PHILIP NELSON, *Liverpool Med.-Chirurg. Journ.*, 1916, xxxvi., p. 51.

THERE is in this paper a discussion of the clinical features of both incomplete and complete division of the nerve, and of the varying effects of its division on the sensation of the hand. A précis of eighteen cases is given with twenty-one figures. The writers summarise the treatment thus:—In cases in which there is complete loss of function—

1. For three months the hand and fingers should be supported on a light cocked-up splint, the muscles being massaged daily and passive movements of the joints carried out. The muscles should also be stimulated by the galvanic current.

2. If recovery in any muscles, even though very slight, can then be discerned, this treatment should be continued, and further, the patient encouraged to attempt voluntary movement; for it is important to remember that the stimulus of voluntary movement is far more potent than that provided by passive movement.

3. If, on the other hand, there is no recovery, the nerve should be explored. We venture to emphasise that the surgeon should have clearly in his mind the conditions he is likely to meet with, and have determined as far as possible beforehand the proper procedure to be adopted. From the neurological standpoint, any traction on the proximal end is most undesirable, since this will certainly cause retrograde changes in the anterior cornual cells.

4. After operation, postural fixation, massage, and passive movement are still urgently required.

5. If, at the end of six to eight months from the date of operation on the nerve, not even the slightest voluntary movement is discernible, then we think the propriety of tendon transplantation should receive careful consideration.

6. If, when the nerve is first examined, eight months or more have elapsed since the date of injury, and complete loss of function is found, then the prognosis as regards recovery is doubtful. The muscles give either no response, or a very feeble contraction to galvanism, and it is not unlikely that the anterior cornual cells themselves have undergone retrograde changes. Tendon transplantation should again be considered. (This paper should be read in the original for many valuable points.)

LEONARD J. KIDD.

APPARENTLY SPONTANEOUS PARALYSIS OF THE ULNAR (186) NERVE, WITH HYPERTROPHY OF THE NERVE TRUNK IN ITS COURSE ALONG THE OLECRANON. (Paralysies du nerf cubital, en apparence spontanées avec hypertrophie du tronc nerveux dans sa traversée olécraniennne.) J. A. SICARD and P. GASTAUD, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1765.

THE writers have observed five cases in soldiers who had not received any wounds or injuries of any kind. During the first stage, which lasted from about two to three weeks, they had pain radiating from the elbow to the upper arm and forearm. During the second stage there was paresis in the region of the hand, and especially of the last two fingers, with sensation of numbness and formication.

In the third stage there were pronounced muscular atrophy (pathological snuff-box), and more or less disturbance of the electrical reactions which might amount to RD.

These ulnar pareses have a relatively long course. The prognosis is favourable.

Their production is due to compression of the nerve at the olecranon by periarticular fibrous formation of rheumatic origin.

In none of the cases was there any history of gonorrhœa, syphilis, or alcoholism.

J. D. ROLLESTON.

THE EFFECT OF PERI-ARTERIAL SYMPATHECTOMY OR OF (187) RESECTION OF A SEGMENT OF AN OBLITERATED ARTERY ON THE VOLUNTARY CONTRACTION OF THE MUSCLES. (Influence de la sympathectomie périartérielle ou de la résection d'un segment artériel oblitéré sur la contraction volontaire des muscles.) R. LERICHE and J. HEITZ, *Compt. Rend. Soc. de Biol.*, 1917, lxxx., 17 Févr., p. 189.

SYMPATHECTOMY, performed in eight cases of reflex paresis or contracture of the Babinski-Froment type, was followed by return of certain muscular movements at the end of twenty-four to thirty-

six hours. Resection of an arterial segment was performed in six wounded subjects who had also lesions of nerve trunks. Here also voluntary movement returned in twenty-four hours. The writers attribute it to the local vaso-dilatation which follows these operations on the peri-arterial sympathetic plexus: the voluntary muscles improve functionally by virtue of their improved blood supply.

LEONARD J. KIDD.

THE ELECTRICAL REACTIONS OF LOCAL HYPOTHERMIA.

(188) (*Sur les réactions électriques d'hypothermie locale.*) H. BORDIER, *Bull. de l'Acad. de Méd.*, 1917, lxxvii., 6 Févr., p. 172.

THE writer has seen a large number of wounded patients during the winter months in whom there existed a reaction of degeneration in the small muscles of the hand or foot, although their motor nerves were in no way injured. There was slow response to galvanism, polar inversion, and a more or less diminished reaction to faradism. These electrical changes can be produced in normal persons by plunging the hand into very cold water for about fifteen minutes. (Bordier refers to the findings of Weiss and other physiologists on the effect of cold on the muscles of animals.) These reactions of local hypothermia are commoner and more marked in the hypothenar than in the thenar muscles, the reason being that when the hand is flexed the former are more exposed to cold than the latter. Bordier has also made the observation that when the frozen hand is submitted to diathermy the electrical reactions become raised to normal almost instantaneously. He holds that this influence of local hypothermia, in inducing a very puzzling RD., ought to be known widely to electrical officers in military hospitals. He found that when this paradoxical condition was present during the winter months it disappeared spontaneously at the end of April.

LEONARD J. KIDD.

"SHELL SHOCK."

NERVOUS DISORDERS DUE TO VIOLENT EXPLOSIONS. (*Sur* (189) *les accidents nerveux déterminés par la déflagration de fortes charges d'explosifs.*) CL. VINCENT, *Rev. Neurol.*, 1916, xxiii., April-May, p. 573.

THREE types of disorder are differentiated—emotional states, states of concussion, and mental disorders. The author admits that a certain amount of overlapping may take place, but roughly he differentiates these states as follows:—The man who is emotionally disturbed does not lose consciousness, is able to look after himself, and can come unaided to the dressing station.

The patient who is concussed is immediately, and for a longer or shorter time, unconscious. The mental patient is neither one thing nor the other, but generally is mentally inert and confused.

In this paper the mental disorders are not discussed, but the cases showing a disorder of mood and the states of concussion were analysed both in regard to their immediate and more remote symptoms. The discussion is reported in full.

D. K. HENDERSON.

CHARACTERISTICS OF THE SO-CALLED "FUNCTIONAL" (190) MOTOR DISORDERS. (*Les caractères des troubles moteurs dits "fonctionnels," et la conduite à tenir à leur égard.*) M. J. BABINSKI, *Rev. Neurol.*, 1916, xxiii., April-May, p. 521.

THE author distinguishes two types of functional motor disorders, those which are easily cured by psycho-therapy, and those in which one is inclined to believe that the patient is opposing recovery. The diagnosis in the first group of cases is based essentially upon the exclusion and absence of all the symptoms forming the positive signs of all other disorders. In the second group of cases, in addition to the persistence of the symptoms, it is necessary to take into consideration the presence of vaso-motor and trophic disorders, persistence during sleep, sensory disturbances, the mental state of the patient, &c., &c.

In addition to the above groups of cases, attention is also drawn to the persistence of tremor in shell-shock cases, and these also the author would term "functional."

If these cases are not curable by psycho-therapy, should one not, as demanded by the Society of Neurology, refuse to these patients discharge or sick leave? The resulting discussion is fully reported.

D. K. HENDERSON.

THE EFFECTS OF HIGH EXPLOSIVES ON THE EAR. J. GORDON (191) WILSON, *Brit. Med. Journ.*, 1917, March 17, p. 353.

"CASES of nerve deafness due to shell concussion" can be divided into three groups:—

1. Those with nerve deafness.
2. Those who have had nerve deafness to a varying degree, and who have the fixed idea that they cannot hear.
3. Malingerers.

This paper only deals with the first group, which may roughly be classified as follows:—

- (i.) Cases of nerve deafness associated with damage to the conducting mechanism.

(ii.) Cases without any visible or demonstrable lesion in the conducting mechanism, although this may have been present at the time of concussion.

(iii.) Cases with destruction of the cochlea and of the semi-circular canals or their nerves.

Over eighty cases were examined, and a few are described here.

Treatment is founded on the following anatomical and physiological basis:—

(a) At the synapses there is structural discontinuity of the nerves.

(b) The results produced by impulses travelling in a nerve depend on the way the fibre ends, and not on any difference in the impulses themselves.

(c) At the synapses different physiological systems come into touch with each other, and so co-ordinate action in diverse systems is possible.

(d) At the synapses there is always a spread of the nerve impulse, and the greater the impulse the greater the spread.

As a result of the high explosive, with sudden increase of pressure in the ear, there occurs a dissolution of the permanent auditory pathway and a spread of the nerve impulse into other adjacent paths. The auditory stimulus no longer reaches its goal and deafness results. Such a dissolution may occur at one or at all the synapses, it may not be complete and a maximal stimulus may be still able to get through. This dissipation of the nerve impulses may help to account for the associated nervous phenomena.

In the totally deaf, either in one or both ears, treatment was as follows:—

1. Tuning forks are applied: (a) to the bone (*e.g.*, mastoid); or (b) through resonators attached to the ear by tube; (c) through the air. In the worst cases the time given to (a), (b), or (c) varies with the amount of deafness.

2. The voice is used: (a) through resonators with tube in ear; (b) through speaking tubes; (c) without any aid.

3. Each period of treatment is very short, as fatigue, headaches, vertigo, sweatings, &c., may be rapidly produced.

4. Carefully graduated physical exercises of short duration and no bendings. Treatment should be twice a day.

5. Ascertain by caloric and rotation tests if the semicircular canals are acting.

Electricity is contraindicated and liable to produce vertigo. The normal stimulus (musical notes or voice) is an adequate stimulus for the nerve, and is the best stimulus. Frequently the field of vision is more contracted on the side having the greater deficiency of hearing. There is usually a diminution all along

the scale both for bone and air conduction, and as the deafness diminishes, there may persist for a long time an inability to grasp intelligently what is said or to retain the memory of it.

A. NINIAN BRUCE.

ARTERIAL TENSION IN THE SUBJECTS OF WAR-DEAFNESS.

(192) (*La tension artérielle chez les sourds de la guerre.*) MARAGE, *Compt. Rend. de l'Acad. des Sci.*, 1917, clxiv., 5 Mars, p. 416.

A STUDY of 185 cases of soldiers rendered deaf by explosions of large shells. All complained of insomnia and severe frontal headaches. The arterial tension was studied by Pachon's sphygmometric oscillometer. Eighty-two per cent. had a tension above the normal; a few had great hypertension, and a small number hypotension. The insomnia nearly always coincided with hypertension, which showed no tendency to subside spontaneously with the lapse of time. The violent headaches appeared to have no relation with the arterial tension: often they lessened with time. Of drugs, valerianate of ammonia gave the best results. But Arsonvalisation always lowered the arterial tension, and Marage regards it as the treatment of choice.

LEONARD J. KIDD.

THE TEST OF TWO VIBRATING TUNING FORKS, OF THE

(193) **SAME NOTE, IN DIAGNOSIS OF FEIGNED UNILATERAL DEAFNESS.** (*Epreuve des diapasons unissonnants appliquée au diagnostic de la surdité unilatérale simulée.*) E. ESCAT, *Presse Médicale*, 1916, Dec. 7, No. 68, p. 562.

THE principle on which Escat's test is based is that "when the two ears of a normal person are exposed simultaneously and respectively to the influence of two sounds of the same tonality and timbre, but of different intensity, the perception of the resultant unison is lateralised entirely in the ear exposed to the louder sound, while its fellow exposed to the feebler sound appears to receive no impression." Escat has had complete satisfaction with the results obtained by his test. It is carried out by two tuning forks of the note A (435 vibrations, second space of treble clef), one of which weighs 305 gm. and vibrates for 180 seconds, while the other weighs only 37 gm. and vibrates for 70 seconds. The former gives far the greater intensity of sound. The patient is seated, with eyes bandaged. The examiner stands in front of him (it is possible to perform the test without bandaging the eyes; the examiner then stands behind patient and out of his field of vision). Suppose patient complains of left deafness. The test has two parts, (1) the test proper, performed thrice,

(2) a counter-test. (A) First time: the smaller fork, vibrating feebly but yet amply enough for a normal ear, is held at 2 cm. distance from left ear. Patient questioned, replies, "I don't hear." (B) Second time: as soon as this reply has been given, the same fork is held 2 cm. from right ear; patient replies, "I hear." (C) Third time: while the small vibrating fork is still held in front of right ear, the larger fork, struck violently against a rubber cushion, is held 2 cm. from left ear: at this moment patient is asked, "On which side do you hear?" Now a really deaf person answers, "On right." But a simulator may answer either (1) "I don't hear on either side," or "I hear nothing," or (2) "I hear on right." In case of (1) the fraud is obvious (the examiner must assure himself at this moment that the smaller fork is really vibrating perceptibly). In case of (2) the matter is easily and decisively settled by the counter-test, performed thus: while the larger fork is kept close to the left ear, the smaller is withdrawn from the right ear: patient is now asked, "On which side do you hear?" To this a really deaf person replies, "I don't hear on either side." But the simulator may reply either (1) "I hear always on the right," or (2) "I don't hear on either side." The first reply, which is far the commoner, proves the fraud. The second reply is rarely given, so much so that Escat (who wrote on this subject as long ago as 1910) has never met with it in his practice. He insists on the need for the most careful attention to the technique, for the least fault on the clinician's part is fatal. He points out that his test is quite distinct from that of Stenger, and also that the principle on which his own test is based is capable of application by other means than vibrating forks, viz., by generators of quite different sounds. (The reasoning by which Escat reaches his various conclusions is given in the paper, but most of it will be clear to the reader of this abstract.)

LEONARD J. KIDD.

SOME FORMS OF FATIGUE AT THE FRONT. (*Quelques formes (194) de la fatigue à l'avant.*) M. LELONG, *Paris méd.*, 1917, vii., p. 66.

THESE consist of lumbago, febrile attacks, hystero-epilepsy, and psychical disturbances such as hallucinations, apathy, and mania.

J. D. ROLLESTON.

PATHOLOGICAL FUGUE STATES OCCURRING BEFORE THE ENEMY. (*Sur quelques cas de fugue pathologique devant l'ennemi.*) B.-I. LOGRE, *Rev. Neurol.*, 1916, xxiii., July, p. 20.

THE author points out the great importance of the study of *fugue* states in the army, owing to the fact that in time of war *fugue*

becomes synonymous with desertion, and is apt to entail either several years' imprisonment or death. Four cases are reported occurring respectively in a case of alcoholism, melancholia, epilepsy, and in an abnormal affective state with phobias and obsessions.

D. K. HENDERSON.

ON SIMULATION. (*De la simulation. La carotte.*) E. BLUM, *Journ. de méd. de Bordeaux*, 1916, lxxvii., p. 274.

AN interesting account of the "carottier," as the "skrimshanker" in the French army is called. The following classification is given of his methods:—

1. Mechanical methods, such as pressure, bruising, and tight bandaging.
2. Chemical and pharmaceutical products.
3. Irritating agents, viz., heat, cold, certain plants, and injection of petroleum or turpentine.
4. Septic agents, *e.g.*, wounds with infected instruments.
5. Voluntary mutilations.
6. Equinism and camptocormia.
7. Simulation of a foreign body in the abdomen.

A description follows of the diseases or symptoms simulated according to the various systems.

J. D. ROLLESTON.

FUNCTIONAL GASTRIC DISTURBANCE IN THE SOLDIER. (197) COLIN M'DOWALL, *Journ. Ment. Sci.*, 1917, lxiii., Jan., p. 76.

THE author has met with many cases of neurasthenic vomiting in soldiers due to emotional disturbance of a direct or indirect nature. The vomiting may, or may not, be associated with food; is not preceded by pain; may occur at night while the patient is in bed; is commonly associated with the stooping posture assumed in doing ward work; is brought on by railway and motor travelling; is stimulated by waterbrash in some cases; produces the sensation of the abdominal contents swaying backwards and forwards; and not infrequently is accompanied by loss of the pharyngeal reflex. The vomiting appears after physical examination to be causeless, and the exact mechanism of the production of symptoms is not clear, and how much is due to vagal stimulation is uncertain.

The main point is that the vomiting is the result of emotional stress, and by understanding your patient, giving him true insight into the production of his symptoms, removing any worrying element, gradually restoring his self-confidence, the offending emotional tone can be controlled or removed. Each case must be treated on its own merits. Several cases are cited as examples.

H. DE M. ALEXANDER.

MENINGITIS.

CASES OF CEREBRO-SPINAL FEVER IN THE ROYAL NAVY—

(198) 1st August 1915 to 31st July 1916. H. D. ROLLESTON, *Lancet*, 1917, i., Jan. 13, p. 54, and *Jour. Roy. Naval Med. Service*, 1917, iii., p. 1.

DURING this period there were 104 cases of cerebro-spinal fever in the Royal Navy as compared with 170 cases during the first year of the war (*v. Review*, 1916, xiv., p. 36). Of these seventy (or 67·3 per cent.) were below 20 years of age. The extremes of age were 15 and 46 years. The average age of the 104 cases was 20·4 years; of the fatal cases, 22·2; and of the recoveries, 19·4 years. Thirty-seven (or 35·6 per cent.) proved fatal, as contrasted with ninety (or 52·9 per cent.) during the first year of the war.

The mode of onset sometimes varied from the common form characterised by fever, malaise, headache, and vomiting. Eight patients, when first discovered, were unconscious; four of these proved fatal. Four cases suggested pneumonia and one appendicitis. Rashes were present in fifty-one (or 49 per cent.); in forty-one it was petechial or hæmorrhagic, and fifteen of these proved fatal. In five cases there was a macular rash, with one death. Although a profuse hæmorrhagic rash is extremely ominous, the occurrence of a rash is not necessarily of grave significance. Herpes was noticed in twenty-one cases (or 20·2 per cent.), in twenty being on the lips; in the remaining case it affected the nasal branch of the ophthalmic division of the fifth nerve. Photophobia was noted in ten cases, conjunctival hæmorrhages in five, strabismus in eight, ptosis in three, nystagmus in one, hemiplegia in one, facial paralysis in one (fatal), dysarthria in one (recovery), and dysphagia in one (recovery). Delirium tremens occurred in two cases, and pericarditis in two, both of which recovered. Synovitis was noted in four cases (two fatal). Mixed infections of the cerebro-spinal fluid were reported in three cases. Only those cases in which meningococci were found in the cerebro-spinal fluid were included here, cases with meningeal symptoms and meningococci in the naso-pharynx only being ruled out. During the acute stage swabs from the naso-pharynx are often negative.

Of ninety-five cases treated with some kind of serum, the mortality was thirty (or 31·6 per cent.). During the first year of the war, out of 105 cases, there was a mortality of sixty-four (or 61 per cent.), and this seems to justify the widely expressed view that the serums employed in this country during the first year of the war were largely deficient in antibodies. Vaccines were given in eight cases, but did not appear to exert any decided beneficial

effect. Serum rashes were mentioned in 22 or 23 per cent. of the cases, and on the average appeared on the tenth day after the first injection of serum.

A. NINIAN BRUCE.

CEREBRO-SPINAL FEVER. R. C. C. CLAY, *Brit. Med. Journ.*, 1917, (199) i, Feb. 24, p. 262.

FIFTY-ONE cases (14 female and 37 male) were admitted to the Alexandra Cerebro-Spinal Fever Hospital, Wigmore, Kent, from July 1915 to December 1916, 14 being soldiers. Of these, 3 were moribund, and died in a few hours. Excluding these, the deaths numbered 14, or 28 per cent. The oldest case was 64 years of age. The average day of disease on admission was the sixth. Twenty-two cases showed no rash, 29 showed one or more types of rashes as follows:—Herpes 10, petechia 15, purpura 6, erythema 3, and macules 1. Pressure erythema was always present. A serum rash developed in 77 per cent. of cases which survived the tenth day; the average date of its appearance was on the tenth day after the first dose of serum. The following complications were seen: temporary insanity 1, arthritis 6, chronic hydrocephalus 4, severe neuritis 1, complete paralysis of the eye 1, parotitis 1, and psoas abscess 1. Polyuria was a very common early symptom.

A. NINIAN BRUCE.

ON THE DIFFERENTIAL DIAGNOSIS BETWEEN TYPHUS AND (200) CEREBRO-SPINAL MENINGITIS. (*Sur le diagnostic différentiel entre le typhus exanthématique et certaines formes hémorragiques de méningite cérébro-spinale.*) V. BABES, *Compt. rend. Soc. de Biol.*, 1916, lxxvii., p. 857.

TOWARDS the end of 1915 and beginning of 1916 there were some cases of typhus in the civil population of Bucharest, while in the barracks several cases were met with presenting many resemblances with typhus. They were not, however, typical, the fever was irregular, less high, and the patients suffered from headache and diarrhoea. A roseola and petechiæ appeared on the second day. Several recovered. Microscopical examination of the skin lesions showed the presence of meningococci in the epithelial layers and intracellular lymphatic spaces.

J. D. ROLLESTON.

A CASE OF SUPPURATIVE MENINGITIS WITH GLYCOSURIA (201) SIMULATING DIABETIC COMA. FRANK E. TAYLOR and W. H. M'KINSTRY, *Lancet*, 1917, cxcii., Feb. 3, p. 182. (1 chart.)

A SOLDIER, aged 36, complained of severe pain in the right side of the chest, cough, and difficulty of breathing. The case was

diagnosed as pleurisy and pneumonia. Three days later twitching of the face was noticed, followed by vomiting, collapse, incontinence, and later coma. The respirations rose to sixty-four, and presented the appearance of air-hunger. The urine was now found to contain a large amount of sugar, but no acetone or diacetic acid. No history of glycosuria could be obtained from the patient's wife. Next day no sugar could be found in the urine, and the percentage of urea was normal. The temperature rose to 107° just before death.

At the autopsy the pleura covering the right lung was covered with a purulent exudation about a quarter of an inch thick. On removing the outer coverings of the brain, pus was found mapping out the sulci on the anterior, superior, posterior, lateral and internal surfaces, and also following the course of the blood vessels. The upper surface of the cerebellum was also covered with pus. Smears and cultures showed both streptococci and *Staphylococcus aureus*.

The glycosuria in this case was considered to be of neurogenous origin.

A. NINIAN BRUCE.

PREVENTION OF CEREBRO-SPINAL FEVER. W. H. PARKES, *Brit. (202) Med. Journ.*, 1917, i., Feb. 24, p. 262.

OWING to the prevalence of cerebro-spinal fever on transports and military camps in New Zealand for fifteen months prior to October 1916, the following measures have been adopted:—All troops before embarkation have the nasopharynx swabbed and examined bacteriologically, only negative cases being allowed to proceed. The contacts are treated by a steam apparatus (*v. Review*, 1916, xiv., p. 464), by which a disinfectant solution is sprayed into the air of a room of 700 cub. ft. capacity, the steam atomizing from a vessel containing 1 per cent. solution of zinc sulphate, of which 1 litre suffices for twenty minutes. Eight carriers are treated for five minutes in the prone position, inhaling the misty air freely through the nose; this temporarily destroys the meningococcus in the nasopharynx. The inhalations are repeated daily for three, four, or five days until the results of the swab examinations are negative. Inhalation rooms are fitted up at the military camps, and also on all transports proceeding to England. During the six months this has been in use, no case has developed on a transport, and the disease is considered to be now under control in New Zealand.

A. NINIAN BRUCE.

- THE APPLICATION OF SURGICAL METHODS TO THE TREATMENT OF CEREBRO-SPINAL MENINGITIS.** H. V. DREW, *Brit. Med. Journ.*, 1917, i., Feb. 17, p. 223.

THE author points out the value of repeated lumbar puncture in cerebro-spinal fever, and suggests that in cases where such treatment is impossible, *e.g.*, those in which the fluid is too purulent or too inspissated to flow at all, and which supply most of the deaths, partial laminectomy might be done for the purpose of direct drainage, and perhaps lavage with saline, in order to establish a flow of spinal fluid which has been interrupted by adhesions.

A. NINIAN BRUCE.

- THE RELATION OF THE TYPE OF COCCUS TO THE TYPE OF DISEASE IN MENINGOCOCCAL MENINGITIS.** ALEX. MILLS KENNEDY and C. C. WORSTER-DROUGHT, *Brit. Med. Journ.*, 1917, i., Feb. 24, p. 261.

GORDON has been able by the method of "absorption of agglutinins" to differentiate the meningococcus into four strains, based upon their immunological characters, and has prepared four immune serums from young rabbits which could be used for the differential diagnosis of the type of meningococcus.

The authors studied twenty-three cases with the view of determining if any difference in the virulence or pathogenic reactions of the four types of coccus could be discovered. Type I. was found in eight cases, all extremely severe, six proving fatal. Type II. was also found in eight cases, all of which recovered, only three being severe. Type III. occurred six times, two proving fatal and four being moderately severe, only one dying. Only one doubtful case, which recovered, belonged to Type IV. Allowance must, however, be made for the different times at which these cases came under treatment.

A. NINIAN BRUCE.

- ON VITAMINES, AMINO-ACIDS, AND OTHER CHEMICAL FACTORS INVOLVED IN THE GROWTH OF THE MENINGOCOCCUS.** D. JORDAN LLOYD, *Journ. of Path. and Bacteriol.*, 1916, xxi., Dec., p. 113.

THE primary cultivation of the meningococcus *in vitro* is only possible in the presence of certain accessory growth factors (the so-called vitamins) present in blood, serum, milk, and other animal fluids, and probably present also in vegetable tissues. These accessory factors are bodies which are moderately heat stable. They are soluble in water and alcohol. They are rapidly adsorbed from solution by filter paper, but do not appear to be

adsorbed by glass-wool. There is a relationship of the inverse order between the amount of amino-acid present in the medium and the amount of vitamine required to stimulate the growth of laboratory strains, and it is therefore suggested that the action of the accessory growth factors is to increase the reaction velocity of the proteolytic metabolism of the meningococcus. The meningococcus, after isolation from the body, gradually develops a change in its metabolism of such a nature that it becomes increasingly independent of a vitamine supply in the medium. Old laboratory strains need no additional vitamine supply if the medium contains an abundant supply of free amino-acids. The main food requirements of the meningococcus are the products obtained by the hydrolysis of a protein, namely, amino-acids. Cultures of the meningococcus must be kept permanently in a moist atmosphere in order to retain their vitality.

A. NINIAN BRUCE.

**ON THE PRESENCE OF AN ACCESSORY FOOD FACTOR IN
(206) THE NASAL SECRETION AND ITS ACTION ON THE
GROWTH OF THE MENINGOCOCCUS AND OTHER
PATHOGENIC BACTERIA. (Preliminary contribution.)**
CRESSWELL SHEARER, *Lancet*, 1917, cxcii., Jan. 13, p. 59.

THE author records experiments which show that in nasal secretion there is present some body which greatly accelerates the growth of the meningococcus on an artificial culture medium. Alone it is incapable of acting as a food or stimulant to the growth of this germ. It is soluble in water, less so in alcohol, and very insoluble in ether. It has great heat-resisting power, being able to resist prolonged boiling for many hours. It is not destroyed by boiling in the presence of strong hydrochloric acid for twelve hours. In addition to the meningococcus it also stimulates the growth of many other pathogenic germs.

A. NINIAN BRUCE.

TETANUS.

**NOTE ON THE INCIDENCE OF TETANUS AMONG WOUNDED
(207) SOLDIERS.** Sir DAVID BRUCE, *Brit. Med. Journ.*, 1917, i., Jan. 27,
p. 118. (1 chart.)

THE object of this paper is to draw attention to the remarkable diminution in the number of cases of tetanus following the introduction of prophylactic injections of antitoxin, and to emphasise the desirability of following up the primary injection by secondary and further prophylactic doses as long as the wound remains suspicious.

A. NINIAN BRUCE.

NOTES ON THREE CASES OF TETANUS. R. I. DOUGLAS and (208) C. H. CORBETT, *Brit. Med. Journ.*, 1916, i., Jan. 27, p. 119.

THE first two cases developed tetanus about *three weeks* after frost-bite, the third followed *one month* after a wound to the left hand. Large and repeated doses of antitetanic serum apparently helped to ultimate recovery. Injections into the limbs were made in most cases below a bandage acting as a constriction to the circulation, and the limb was allowed to retain the antitoxin for five or six minutes. Twitchings in the limb were certainly lessened, and probably the spread to a more generalised spasm frequently averted by this treatment.

A. NINIAN BRUCE.

LATE TETANUS FOLLOWING PNEUMOCOCCAL INFECTION.

(209) (*Sur un cas de tétanos tardif à la suite d'une infection pneumococcique.*) MACLAUD and E. LÉVY, *Paris méd.*, 1917, vii., p. 163.

A SENEGALESE soldier, aged 25, was wounded on 2nd August, and received 10 c.c. of tetanus antitoxin the following day. On 12th September, a week after the wound had completely cicatrised, he developed pneumonia, and the following day generalised tetanus. Recovery took place without serum treatment or carbolic acid.

The writers regard intercurrent disease as a predisposing cause of the late tetanus, the medical infection acting like a trauma on the spores of *B. tetani*.

J. D. ROLLESTON.

A CASE OF RECURRENT TETANUS. A. M. WESTWATER, *Brit. Med. Journ.*, 1917, i., March 24, p. 394.

A SOLDIER was wounded over the right scapula by shrapnel, and received a prophylactic dose of antitetanic serum the same day. Symptoms of tetanus arose on the *sixteenth* day. Between this and the twenty-sixth day he received 27,000 units of antitetanic serum. On the fifty-fourth and on the sixty-fourth days he received a prophylactic dose of 500 units.

The second attack occurred on the sixty-seventh day, *i.e.*, after *forty-two* days of apparently complete recovery from the first attack. Between that and the seventy-fourth day, when he died, he received 21,000 units intrathecally.

A. NINIAN BRUCE.

LATE TETANUS. JAMES MILLER, *Brit. Med. Journ.*, 1917, i., Feb. 17, (211) p. 223.

A SOLDIER who was wounded on 22nd October 1916 received a prophylactic injection the same day, and again after eight days. On 22nd November there was slight rigidity of the left arm. On

8th December the wound was completely healed, and he returned to duty seven days later. Pain in the head developed on 5th January 1917, followed six days later by retraction of the head, trismus, &c. He was given 3,000 units of serum intrathecally, and 1,500 units intramuscularly, repeated next day, but died from pneumonia. The incubation period was thus about *seventy-five* days.

A. NINIAN BRUCE.

**DELAYED TETANUS IN CONNECTION WITH INJURIES TO
(212) BONE NOT PRESENTING OBVIOUS SIGNS OF SEPSIS.**

M. FOSTER, *Brit. Med. Journ.*, 1917, i., Feb. 10, p. 189.

THREE cases are recorded, all of which had sustained gunshot wounds with fracture, and which developed tetanus later. The periods of incubation were 146, 106, and 86 days respectively. So far as could be ascertained, a primary prophylactic dose had been given in every case. The exciting cause of the symptoms of two of the cases appeared to have been the direct injury of a fall, which had lighted up a quiescent focus. All recovered with intrathecal and intramuscular injections.

A. NINIAN BRUCE.

**THE TREATMENT OF TETANUS BY LARGE DOSES OF ANTI-
(213) TETANIC SERUM COMBINED WITH CARBOLIC ACID.**

(Le traitement du tétanos par le sérum anti-tétanique à haute dose associé à l'acide phénique.) J. COSTE, *Journ. de méd. de Bordeaux*, 1916, lxxxvii., p. 282.

A RECORD of six cases of generalised tetanus in soldiers, none of whom had received a preventive injection of anti-tetanic serum. All recovered. No patient received less than 60 c.c., or more than 160 c.c. of serum in all. Subcutaneous injections were most frequently used. The strength of the carbolic acid solution was 1 in 200. Doses of 0.30 to 0.35 ggm. a day were given.

J. D. ROLLESTON.

**ON CASES OF POST-SERIC TETANUS. (Sur les tétanos post-
(214) sériques.)** AUGUSTE LUMIÈRE, *Ann. de l'Institut. Pasteur*, 1917, xxxi., Janvier, p. 19.

A STUDY of fifty-four cases of tetanus following anti-tetanic serum injections. Conclusions:—

1. Preventive injections of anti-tetanic serum have not an absolute and unlimited prophylactic action.

2. The duration of the absolute immunity conferred by the serum cannot be fixed; it depends on the relative proportions of the toxin and the preventive serum which are contending in the system.

3. Cases of post-seric tetanus appear to be due to the following two principal causes:—

- (a) Excessive secretion of the toxin in the wounds, out of proportion with the dose of serum injected (early post-seric tetanus);
- (b) Liberation of the spores of tetanus, which are in latent activity in the tissues, by means of surgical operation or trauma when the activity of the antitoxin is exhausted (late post-seric tetanus).

4. Early post-seric tetanus can in most cases be prevented by laying bare the infected wounds and careful removal of any foreign bodies they may contain, and by free drainage and repetition of injections of serum once or oftener.

5. Late post-seric tetanus is also preventable, in more than half the cases, by injecting a new dose of serum in every instance of secondary operation on the wounds.

6. Preventive sero-therapy sometimes imparts peculiar characters to post-seric tetanus, in that it alters more or less the symptomatology and clinical course of the disease.

7. In some cases of post-seric tetanus (15 out of 54) injections of antitoxin have prevented fixation of the poison in the central nervous system, its action being confined to the motor nerves of the wounded limb. These cases of localised tetanus without trismus are much less grave than the other forms.

8. In other cases (13 out of 54) there is only partial protection of the bulbo-spinal centres: there is a late or partial trismus with local contracture: the prognosis is then less favourable.

9. The prognosis is worst in those cases (26 out of 54) in which antitoxin has failed to protect the central nervous system, *i.e.*, cases which show trismus from the outset.

The treatment of post-seric tetanus consists in as early as possible use of large doses of the serum. At present there is no absolutely curative treatment: we must combat the symptoms. We can do little for the permanent contractures, but the paroxysmal spasms can be treated by stupefants, chloral, morphine, injections of sulphate of magnesia or persulphate of soda: the last-named appears to be the remedy of choice on account of its efficacy and feeble toxicity.

LEONARD J. KIDD.

THE PLACE OF CURARE IN THE TREATMENT OF TETANUS.

(215) JOHN S. M'ARDLE, *Dublin Journ. of Med. Sci.*, 1917, Ser. 3, April, p. 239.

So long ago as 1885 the writer recorded a case of tetanus in which two-third grain doses of urari every fifth hour resulted in cure: the contracted muscles relaxed in from six to ten minutes after

administration, and there was very rapid, tumultuous action of heart, cyanosis, laboured breathing, and dilated pupils. Later he tried wourali; recovery followed, preceded by alarming symptoms; pilocarpin was also given hypodermically. Then urari, in doses of gr. $\frac{1}{10}$ to gr. $\frac{1}{8}$ often proved effectual. All this was before the days of serum treatment of tetanus. Under these drugs the unstriated muscles functionate normally, so that the patient's strength is maintained while his tissues destroy the circulating tetanus toxin. In the treatment of tetanus, curare is given hypodermically in doses of gr. $\frac{1}{32}$ to gr. $\frac{1}{8}$, or curarine similarly in doses of gr. $\frac{1}{200}$ to gr. $\frac{1}{40}$.

The writer's present practice is to treat cases of tetanus by early serum injection with curarine hypodermically on the slightest sign of local or general spasm: he has thus had many gratifying successes. He now never sees a soil-infected wound without injecting antitoxin, and he always has curare tablets at hand. He advises that the antitoxin should be injected into the nerve supplying the affected part, so as to intercept the toxin on its way to the central nervous system. The rapidity of action of curare is shown in a seemingly desperate case, quoted by the writer, in which within three minutes of a hypodermic injection of gr. $\frac{1}{2}$ the spasms ceased. In this case the dose had to be repeated every sixth hour for three days.

LEONARD J. KIDD.

**ON THE PASSIVE IMMUNITY CONFERRED BY A PRO-
(216) PHYLACTIONIC DOSE OF ANTITETANIC SERUM.** A. T.
MACCONKEY and ANNIE HOMER, *Lancet*, 1917, i., Feb. 17, p. 259.

A NUMBER of experiments upon guinea-pigs are recorded, which show that they exhibit extreme differences in their susceptibility to tetanus toxin, and that to obtain passive immunity of long duration enormous doses must be administered.

It must therefore be considered whether, after giving a prophylactic dose of tetanus antitoxin, further doses should be given to keep up the immunity (in favour of which there is a large volume of opinion), or whether it is not better, after giving the prophylactic dose, to simply keep a careful watch for the first signs of incipient tetanus, and then at once treat the case energetically. Possibly this latter method may ultimately prove best.

A. NINIAN BRUCE.

**COMPARATIVE VALUE OF THE METHODS OF TREATING
(217) TETANUS.** C. L. GIBSON, *Amer. Journ. Med. Sci.*, 1916, clii.,
Dec., p. 781.

EIGHT cases are described. The first case occurred after operation for ovarian cyst and proved fatal. During the Civil War in

America the mortality from tetanus was 89.3 per cent., and during the Franco-Prussian War 90 per cent. The paper includes quotations about treatment from numerous other sources.

A. NINIAN BRUCE.

"TRENCH FOOT."

THE IMPORTANCE OF THE EARLY PROPHYLACTIC IN- (218) JECTION OF ANTI-TETANIC SERUM IN "TRENCH FOOT."

Sir DAVID BRUCE, *Brit. Med. Journ.*, 1917, Jan. 13, p. 48.

THE author states that fifteen cases of tetanus caused by "trench foot" have been recently reported to him, but full reports had only been received in eight. Of these only two recovered. A prophylactic injection was not given in any of them. The average duration of the disease was only 2.5 days.

Every case of trench foot should receive a prophylactic injection of anti-tetanic serum, repeated every seventh day until the wounds are clean.

A. NINIAN BRUCE.

A NOTE ON THE CAUSE AND PREVENTION OF TRENCH (219) FOOT. C. NEPEAN LONGRIDGE, *Lancet*, 1917, i, Jan. 13, p. 62.

THE lesions of trench foot are indolent, slow to heal, prone to break down, and the scar tissue is weak and ulcerates easily. "The whole picture reminds me irresistibly of the trophic sores appertaining to diseases of the central nervous system," and "is an affection of a trophic nature."

According to A. E. Baines, the brain and cord are the seat of electrical generation, and the current so generated is carried to the tissues by the nerves, which are nothing but insulated cables. The dominant factor in causing trench foot is wet. The electrical resistance of the skin is enormously diminished when the skin is wet, and a gradual leakage of electricity to earth thus takes place from the feet. As electricity tends to leak away from a point in preference to a plane surface, the toes become most affected. Five experiments are described which the author thinks afford evidence that trench foot is probably caused by long-continued leakage of electricity from the feet. The best treatment is thorough greasing of the feet with dielectric oil. Rubber boots tend to make the feet sodden, and thus lose their electrical resistance.

A. NINIAN BRUCE.

ON THE ORIGIN OF ELECTRIC CURRENTS LED OFF FROM
(220) **THE HUMAN BODY, ESPECIALLY IN RELATION TO**
"NERVE-LEAKS." W. M. BAYLISS, *Brit. Med. Journ.*, 1917,
March 24, p. 387.

CURRENTS led off from various parts of the body by metallic electrodes are due to inequalities in the electrodes, together with differences in the activity of the skin glands. Neither static charges nor induction plays any part. "Nerve-leaks" are merely places where the skin is moist, and they give no indication of lesions in the nerve centres. The view that "neuro-electricity" is generated in the brain and escapes from nerves owing to breakdown of insulation, is devoid of evidence and contrary to the knowledge we possess of physiological processes.

Professor Bayliss examined Baines' "dielectric oil," and comes to the conclusion that it is ordinary liquid paraffin; that it does not pass through the skin, and cannot reach any nerve or other internal tissue.

Treatment of open wounds by liquid paraffin has some justification in excluding air and perhaps bacterial infection, but it is not new. The results obtained have no insulating properties, and the "dielectric" has no superiority in this respect over commercial samples (*v. supra*).
A. NINIAN BRUCE.

BERI-BERI.

AN OUTBREAK OF BERI-BERI IN THE "EMPRESS OF ASIA."
(221) PERCY B. EGAN, *Journ. Royal Naval Med. Service*, 1917, iii., April,
pp. 195-201.

DURING the period from 5th August to 31st December 1914, 38 cases of beri-beri occurred on board the ship, 5 of which terminated fatally. Thirty-four of the cases occurred among Chinese engine-room ratings, 3 occurred amongst Chinese seamen, and the remaining case was in a European, an engineer officer. The outbreak coincided with the issue of a fresh supply of polished rice, and disappeared with the issue of a fresh supply of unpolished rice. Two distinct types of case were present; the *milder* type began with weakness in the legs, the gait was slow, uncertain, and swaying, with complete inability to increase the speed, and usually slight œdema of the feet. The knee reflexes were in all cases lost. None developed cardiac symptoms, and all the 31 cases in this group did well. The *cardiac* type began with pain in the chest, rapid pulse, and "cantering" rhythm. Weakness of the legs was rarely complained about. Of the 7 cases of this type, 5 died, the illness lasting under three days from the time the patients were first seen.

In two cases fever was present at the commencement of the illness. As all the cases occurred in batches of two, three, or four, and as it was noted that in a few the patients had been sleeping together, the cases were isolated.

A. NINIAN BRUCE.

PSYCHIATRY.

FUNDAMENTAL POINTS OF THE MENTAL EXAMINATION IN
(222) **THE AFRICAN BATTALIONS, AND SPECIAL GROUPS IN**
TIME OF WAR. (Les bases de l'expertise mentale dans les
bataillons d'Afrique et les groupes spéciaux en temps de guerre.)
POROT, *Rev. Neurol.*, 1916, xxiii., July, p. 24.

In the African battalions and in the special groups (reservists and territorials) there are quite a number of persons who are either mentally enfeebled, or who have previously been in asylums. In time of war one should be chary about discharging such individuals, as they are capable of performing certain essential, more or less mechanical, duties. For instance they can carry a sack, or handle a pick, and provided the individual is physically strong enough, there is no reason why a dement, or a mental defective, or a hypomanic patient should not be so employed. The discipline and restraint of a military life has sometimes a really beneficial effect on hypomanics.

General paralytics, chronic confused states with an element of dementia, chronic agitated delirious states, acute manias and melancholias are, of course, quite unfit for service. But discharge is a solution which should rarely have to be employed, as a good many of the above either recover sufficiently to be sent back to their regiments, or else are committed to asylums. The degree of responsibility of these individuals when guilty of wrong-doing or insubordination is also discussed.

D. K. HENDERSON.

THE STUDY OF MENTAL DISEASES IN SOLDIERS DURING
(223) **THE PRESENT WAR.** (Contribution à l'étude des maladies
mentales chez les militaires pendant la guerre actuelle.) E.
MONTEBAULT, *Thèses de Paris*, 1916-17, No. 15.

THE thesis is based on the study of cases of mental disease admitted to the Navarre Asylum from 2nd August 1914 to 30th October 1916.

The writer's conclusions are as follows:—

1. War does not create any special psychoses.
2. In the present war post-traumatic psychoses, due to the violence and power of engines of destruction, are extremely frequent.

3. Contrary to what was observed in 1870, melancholic forms of insanity have been more numerous than cases of maniacal excitement.

4. Most of the military patients were predisposed to insanity, but many of them would probably have remained all their life free from symptoms but for the present war.

Among the causes which have given rise to their mental disturbances, trauma should take the first place; psychical trauma appears to be much the most frequent, while lesions of the nerve centres are relatively rare.

Exhaustion and fatigue which act like an auto-intoxication are next most important. Then come intoxications and infectious diseases.

5. The symptoms of insanity in the military patients have almost all a war colouring which is shown (*a*) in ideas of unworthiness, guilt, and expiation, (*b*) in ideas of persecution and auditory hallucinations, (*c*) in exalted ideas.

6. The civilian patients sometimes show a war delirium, analogous to that of the mobilised.

7. The prognosis of the psychoses caused by the war is favourable as a rule. The proportion of curable cases is about 80 per cent. The duration of the disease is shorter than that of psychoses seen in the civilian population.

8. The curability and rapidity of the cure of these psychoses are due (*a*) to suppression of the determining cause; (*b*) to the age and physical vigour of the patient; (*c*) to rational treatment (isolation) applied as soon as the mental disturbance appears.

J. D. ROLLESTON.

MENTAL DISABILITIES FOR WAR SERVICE. Sir GEORGE (224) SAVAGE, *Journ. Ment. Sci.*, 1916, Oct., p. 653.

It is at times worth running some risk with mental patients: a complete change in mode of life may turn a hypochondriac into a useful worker; but, in taking risks, one must see on whom the risk falls. Some patients might serve as subordinates, but not in positions of responsibility.

Any young man with direct insane inheritance, and who has had a recent (within a few years) attack requiring detention should not enlist, though some of these cases may be fit for home or munition work.

Folie du doute may arise at this time in many young men who have suffered from previous nervous or mental disorders.

Cases of shell shock should not return to service under six months, as relapse is frequent.

Service may turn very suspicious men into paranoiacs.

The author has met a form of functional epilepsy in several cases of men who, as the result of psychic strain or shock, lose consciousness for short periods, and yet when removed from strain they recover, but relapse if sent back to duty.

Cases of *petit mal*, confirmed somnambulism, men with hallucinations, and most mental defectives should be rejected. The writer passed one defective who had been an incurable liar and pilferer while at school.

A man who had previously been in a railway accident and who had a horror of railway travelling ever since, was rejected; as also were two men who from adolescence had been unable to micturate except in private, and others have had to leave the army on account of this latter obsession.

No man with a clear history of symptoms of nerve degeneration related to syphilis should be allowed to enter the army, but there are some whose symptoms are recent, and who after treatment may be allowed to enlist.

H. DE M. ALEXANDER.

MENTAL DISORDERS IN CIVILIANS ARISING IN CONNECTION WITH THE WAR. R. PERCY SMITH, *Proc. Roy. Soc. Med.*, 1916 (Sect. of Psychiat.), x., pp. 1-20.

THE emotional excitement incidental to the onset of the war, the effect of anxiety regarding relatives in the Services, financial disabilities, excessive war work, unaccustomed occupations, the possession of a foreign or German name, coast bombardment, airship and aeroplane raids, the fear of submarine attacks during a voyage, have all in the author's experience induced mental disorders in civilians; but in the great majority of such cases heredity, a previous attack, or some predisposing instability was present. On the other hand, in all probability in many cases tendencies which might have led to the development of neuroses or psycho-neuroses have been "sublimated" by useful work, and the final effect may be a strengthening of the mental constitution of the nation.

H. DE M. ALEXANDER.

GENERAL.

THE ANKLE JERKS OF A SHIP'S COMPANY. HILDRED CARLILL, (226) *Journ. Roy. Naval Med. Service*, 1917, Jan.

THE ankle jerks of 1,051 men were examined. The age of the men was 16 to 50. In no case did the men complain of anything; they considered themselves healthy. The examiner was the same for each case. Total absence of one or both ankle jerks was

detected in 12 cases. Forty-nine of the men required reinforcement before the jerk could be demonstrated. Of the 12 cases in whom the jerk was absent, 2 were instances of former infantile paralysis; 2 had signs of sciatic neuritis; 1 was a case of tabes dorsalis, confirmed by examination of the serum and cerebrospinal fluid. His knee jerks were absent also. In 4 cases a diagnosis of tabes dorsalis was considered probable: of these both jerks were absent in 2 cases and 1 only was absent in 2 cases. Of the remaining 3 men, the diagnosis in 2 was undetermined. One ankle jerk only was absent in them. In the last case there was absence of both knee jerks and ankle jerks, but no other abnormality was detected. In none of the twelve men was the absence of the ankle jerks unassociated with any other abnormality.

In 1,039 men the ankle jerks were normal, and emphasis is laid on the extreme importance of the absence of this reflex as a sign of disease.

AUTHOR'S ABSTRACT.

THREE CASES OF PALPEBRO-OCULAR HERPES FOLLOWING (227) ANTI-TYPHOID VACCINATION. (*Trois observations d'herpès palpébro-oculaire consécutif à la vaccination anti-typhique.*)
GLOAGEN, *Annales d'oculistique*, 1917, cliv, Jan., p. 48.

THREE cases in young soldiers. In the first case the two first injections of Tab vaccine, $1\frac{1}{2}$ c.c. and 2 c.c., given on 2nd and 9th June respectively, were borne perfectly; the third, of 2 c.c., given on 16th June, was followed next day by lassitude, fever (40° C.), headache, and pain in the back, for which lumbar puncture was done; on the same day an intense naso-labial herpes appeared, chiefly on right side. Three days later the right eye reddened and became photophobic. On the 23rd June herpetic keratitis was seen: recovery in eighteen days, but with a persisting fine corneal opacity. In the second case the fourth injection of 2 c.c., three weeks after the first, was followed next day by an extensive, chiefly right-sided, naso-labial herpes. When seen in the clinic three days later, there was a particularly marked palpebral herpes, especially of the upper lid, and a herpetic keratitis involving almost the whole cornea. Under a bandage, atropine, and asepsis, recovery followed in twenty-five days, but a slight corneal opacity persisted and reduced vision to half. The third case was very acute; after the first injection there was severe headache, fever, and dorso-lumbar pains, coming on in the course of a few hours. During the same night naso-labial herpes, and next day photophobia on left side. Three days after the injection the left eye showed herpetic involvement of almost the whole cornea and disseminated islets of herpes on both lids. Recovery only on forty-third day. This patient received only $1\frac{1}{2}$ c.c.

LEONARD J. KIDD.

POST-SCARLATINAL HEMIPLEGIA. (*Hémiplégie post-scarlatineuse.*) L. LORTAT-JACOB and R. OPPENHEIM, *Bull. et mém. soc. méd. Hôp. de Paris*, 1916, xl., p. 1746, and *Progrès méd.*, 1916, p. 213.

A SOLDIER, aged 35, contracted scarlet fever in August 1915. No information was available as to the gravity of the attack, or the occurrence of complications. In October he developed symptoms of mental confusion, and in November, when he appeared to be cured, complete left hemiplegia suddenly occurred, followed by contracture.

Wassermann's reaction was negative. The writers attribute the hemiplegia to cerebral arteritis, and the psychical symptoms to disturbance of the cerebral circulation to which the patient was predisposed by alcoholism. The paper contains a review of the literature, including the articles by Rolleston (*v. Review*, 1908, vi., p. 530), Gouget and Pellissier (*ibid.*, 1909, vii., p. 545), Neurath (*ibid.*, 1912, x., p. 80), and Savy and Fabre (*ibid.*, 1913, xi., p. 436).

J. D. ROLLESTON.

SYPHILIS AFTER TWO YEARS OF WAR. (*La syphilis après deux ans de guerre.*) GAUCHER and BIZARD, *Paris méd.*, 1917, vii., p. 54.

WHEREAS during the first sixteen months of the war (August 1914 to December 1915), syphilis had increased by more than a third (*v. Review*, 1916, xiv., p. 478), during the following eight months it has increased by more than a half, and almost by two-thirds according to the writers' observations. In the civilian population there was an unusually high incidence both among the very young and those advanced in life. In the military population, the youngest class was relatively the least affected, two-thirds of the patients consisting of men between 25 and 35, a large proportion of whom were married. More than two-thirds of the female patients were married, and many of these had noted the first signs of syphilis some weeks after the visit of their husbands home on leave.

The writers insist on the necessity not only of enlightening the public, and especially soldiers, on the dangers of venereal disease, but also of giving every medical man a training in syphilology. Several examples are quoted of syphilis being mistaken by the practitioner for psoriasis, herpes or tonsillitis.

J. D. ROLLESTON.

THE HISTORY OF TWO CASES OF PROLONGED SLEEP. THE (230) VARIETIES OF LETHARGIC STATES. (*Histoire de deux dormeurs. Variété des états léthargiques.*) H. VERGER, *Gaz. Hebd. des Sci. Méd. de Bordeaux*, 1917, xxxviii., Jan. 28, p. 11.

PROFESSOR VERGER has recently had under his care two cases of prolonged sleep which differed markedly from each other in many respects:—

Case 1 was a lyrical artist of 31, who was in excellent health at the outbreak of war; no known history of any nervous attacks. On 6th September 1914, during a battle, he disappeared from his regiment, and no one knows what happened to him. At any rate he received no wound, for four days later he was taken to hospital with a diagnosis of traumatic aphasia. It is now known that even then he was in the same state of deep sleep in which he was when nearly two years later he came under Verger's care. In 1914 he could walk without waking, if held up under the arms. During the month he was under Verger's care his sleep was exactly like natural sleep, except that he could not be wakened; at times he even snored. The eyelids were closed, with a constant tremor; mouth closed; face of natural colour. Regular abdominal respiration, as quick as 20 to 24; pulse regular and rapid, 96 to 110; arterial tension gave by Pachon's instrument 17 for maxima and 7 for minima. Temperature varied from 36.5° to 37.1° C. The whole body was relaxed, but without loss of tonus or tendency to catalepsy. He made the normal movements of a sleeper, lay in dorsal decubitus, and quickly returned to it when placed on his side. Sensory stimuli, *e.g.*, pricks, pinchings, tickling, provoked feeble defensive movements, but had no effect on his sleep. Light and sounds were absolutely without effect on him. All tendon jerks and skin reflexes normal. Pupils could not be examined owing to spasmodic upward turning of eyes on opening of lids. All organic functions normal; swallows liquids easily; micturition and defæcation at regular intervals; never any erections or emissions. He remains remarkably well covered, with very marked relief of muscle. Urine normal; 20 gm. of urea, 5.85 of chlorides, and 1.80 of phosphates in twenty-four hours. Patient never uttered a sound, and never assumed attitudes as if he were dreaming. All attempts to waken him failed; chloroform merely made him vomit; even intense electrical stimulation produced merely defensive movements, and he went on sleeping peacefully. He left hospital after a month's stay, and has not been heard of: probably he is still asleep.

Case 2 is of quite a different kind of lethargy. A man of 28, of unknown antecedents, had typhoid fever in August 1915 while at the Dardanelles, and was sent home on 21st December 1915.

He was received into a hospital in a state of profound prostration, speechless, with clenched teeth and closed eyes. Next day no reactions to sensory stimuli. Fever gone, pulse 72; had to be fed artificially; came under Verger's care on 9th February 1916. He looks like an ordinary sleeping man, but his eyes are closed, with a perpetual tremor of lids, clenched teeth, facial expression calm, and limbs completely relaxed, though there is a slight tendency to maintain them in postures passively induced. Unlike case 1, he remains motionless in any position in which he is put, and does not react to any sensory stimulus. Respiration slow, 10 to 12, of feeble amplitude, commonly regular; but on several occasions was of Cheyne-Stokes type. Pulse 75 to 90. Temperature varied from 36.4° to 37.5° C. Arterial tension by Pachon, maxima 10, minima 6, with maximal amplitudes of only two divisions. The tendon jerks, at first feeble, very quickly disappeared. Alimentation was difficult owing to impossibility of opening his clenched jaws; fed by nasal tube and behind molar teeth. Vomited frequently; this was combated by varying his diet. Micturition at intervals; enemata needed. Undigested food often in fæces. Patient remained in this state for seven months (till September 1916), but wasted progressively and became a living skeleton; all the pelvic and limb bones could be felt easily, and the muscles felt like elastic bands, but continued to react normally to mechanical and electrical stimulation. He looked more like a corpse than a sleeping man. All the vital functions became extremely feeble. He had a profuse diarrhœa, and then his sleep ended: he opened his eyes. At the moment of waking his respiration quickened to 20, pulse to 84; very feeble. During the night he began to speak, asked for rum, and complained of coldness of head and buttocks. During next day was in complete collapse, with a rectal temperature of 36° C. Purpuric streaks could be produced on abdomen and thorax; his face retained its colour. He showed no surprise on waking, and spoke only to ask for rum and coffee, which he drank easily. He answered questions only in monosyllables. Death on that night (2nd September 1916). *Necropsy*.—Nothing but total loss of fat, intestinal retraction due to inanition, and a remarkable smallness of the heart.

Professor Verger contrasts these two interesting cases. The first he regards as one of hysterical lethargy. The second case showed a slowing of all organic functions, with total suppression of cerebral functions, a stuporous state pushed to extremes, or one resembling hibernation of certain animals. Probably a toxic cause was at work, but lack of time prevented proper studies by Verger on this point. The diarrhœa which immediately preceded the waking from the prolonged sleep was possibly a sort of toxic

discharge. From this moment death was seen to be inevitable; for the circulation, just enough for a sleeping man, could not keep alive a waking one. Hence the fatal terminal collapse.

LEONARD J. KIDD.

AURAL REACTIONS IN AVIATORS DURING FLIGHTS. (Les (231) *réactions de l'oreille chez les aviateurs pendant les vols.*)

P. LACROIX, *Bull. de l'Acad. de Med.*, 1917, lxxvii., Jan. 16, p. 94.

LACROIX finds from examination of a large number of aviators during flights of 500 to 1,800 metres, and from personal experiences, that vertigo does not occur during normal aviation, but that there is always some disturbance of hearing, such as tinnitus and intermittent deafness due to the wind and irregularities of pressure on the tympanum in the successive strata of the atmosphere traversed. These disturbances are lessened, almost automatically, by movements of deglutition. The aural symptoms usually pass off soon after reaching ground. They appear to be harmless to the normal ear, but probably an abnormal ear cannot so easily correct them. The candidates for an aviator-pilot certificate ought therefore to have normal middle and internal ears.

LEONARD J. KIDD.

THOMSEN'S DISEASE. (*Maladie de Thomsen.*) L. LORTAT-JACOB and (232) A. SÉZARY, *Rev. Neurol.*, 1916, xxiii., July, p. 15.

A TYPICAL case in a young soldier, 21 years old.

D. K. HENDERSON.

Reviews

AN EMPEROR'S MADNESS, OR NATIONAL ABERRATION?

(233) ERNESTO LUGARO, Professor Extraordinary of Neuropathy and Psychiatry in the University of Modena. Translated by W. N. ROBINSON, M.D. Pp. v+135. George Routledge & Sons, Ltd., London. 1916. Pr. 2s. 6d. net.

WE have already drawn attention to this important criticism of Professor Lugaro of the theory held by some alienists that the cause of the war is to be found in the personal psychology of the two emperors and especially Wilhelm II. (*v. Review*, 1916, xiv., p. 4). Dr Robinson has done good service by translating this into English, and thus making it available to a still larger circle of readers. It is a most telling indictment of Germany closely reasoned out with great skill, and worthy of the closest attention.

In our previous reference we described fully its scope, but the following quotations are also worthy of repetition:—"It is well

known that the psychology of the crowd is inexorably levelling, but no one would have dreamt that the common level could have fallen so low, and one passes from wonder to wonder, listening to what is said by the representative men of Germany, become more loquacious than usual as if wishing to assume their part of the responsibility for the policy which has led to the catastrophe of to-day. Statesmen shriek as unreasonably as the most ignorant, generals make proclamations in the style of a camp-follower, princes exalt fisticuffs and bites, poets sing hate and rage, the Chancellor confesses openly his rôle of secret disturber of the peace, and abuses the enemy nations like a drunken prostitute, university professors endeavour to justify the war, the massacres and the vandalisms, repeating hypocritically miserable excuses which are enough to make a cat laugh, and extol their motives at the same time." "‘Useful cruelty’ makes a pair with the ‘necessity’ of the Chancellor-philosopher; it is the necessity—to conquer, a necessity altogether German, absolutely forbidden to the adversaries." "At bottom this people which aspires to dominate the world is more made to obey than to command." "Does no one recognise the cool courage of civilised men, which does not break out in the bellowings of wild beasts, but prepares an inflexible resistance and just victory?" "Germany has behaved like a gamester, who sits down to the table without money, ready to pocket it if he wins, prepared to stab his adversary and rob him if he loses. Megalomania and violence go hand in hand in perfect accord." "And we must not forget the internal enemies, the neutralists of yesterday, to-day hidden or masked, who to-morrow with the pretext of peace, under the mantle of commerce or of internationalism, would let the danger grow again." "All the civilised nations would willingly see Germany intent on disinterested study and peaceful labour, collaborating in the common progress; but meanwhile they wish to be freed, by good will or by force, from every pretence of hegemony."

MEDICAL DISEASES OF THE WAR. ARTHUR F. HURST. Pp. 151.
(234) Edward Arnold, London. 1917. Pr. 6s. net.

A SHORT account is given here of some of the principal diseases associated with the war as observed by Dr Hurst, and amplified by a study of the literature. The first forty pages are devoted to functional nervous diseases, and then follow chapters upon dysentery in its different varieties, amœbic hepatitis and hepatic abscess, trench fevers, paratyphoid fever, epidemic jaundice, beri-beri, soldier's heart, war nephritis, and gas-poisoning.

The first chapter upon functional nervous disease presents a good general description of the various symptoms most commonly met. The importance of early diagnosis is emphasised, as well as

many other points, such as the presence of cured patients in the same ward with such cases so as to create, as it were, an atmosphere of recovery, &c. Special attention is directed to hypnotism, which the author appears to have made much use of with considerable success. In reply to the criticism that "hypnotism treats symptoms without dealing with the underlying abnormal condition of the nervous system," he states that while "this may be true in non-traumatic cases of hysteria seen in civil life, it does not apply to the hysterical manifestations which occur in soldiers. The symptoms have generally been produced by some quite exceptional circumstance, such as the explosion of a big shell, in spite of the fact that the individual's nervous system was either normal or somewhat exhausted as a result of the strain and stress of war."

The "soldier's heart" is often only one manifestation of the nervous exhaustion or neurasthenia which results from the combined effects of physical fatigue, mental strain, and toxæmia. In a small proportion of cases the intoxication is due to the excessive activity of the ductless glands, resulting from the nervous strain of active service. As over-activity of the thyroid can be detected more easily than that of other glands, there has been a tendency to regard such cases as examples of simple hyperthyroidism, but the condition is really a highly complex one, and might perhaps be more accurately described as a "vegetative neurosis."

Some interesting cases of beri-beri from Lemnos, associated with epidemic jaundice, are described, and the opinion is expressed that the latter infection was as important a factor as food deficiency, though this does not seem to have been the case in Mesopotamia.

The other chapters upon dysentery, trench fevers, &c., are all good. They contain much practical information, and give excellent summaries of our present knowledge of these diseases. The book ought to prove of considerable value to those who have to deal with such cases. Trench foot, cerebro-spinal fever, and tetanus are not mentioned. References to the literature are given at the end of each chapter, and there is an index.

WAR-SHOCK. *The psycho-neuroses in war psychology and treatment.* (235) M. D. EDER. Pp. 154. William Heinemann, London. 1917. Pr. 5s. net.

THE author describes here the first hundred consecutive cases of psycho-neuroses which came under his care in Malta, the psychoses being excluded. Of these 30 possessed a pre-war history (family or personal); 70 showed no such history and thus come under his category of "war-shock." He follows Freud in dividing hysteria into two groups *conversion-hysteria*, where the mental affection is

converted into its physical equivalent, and includes the affections of the senses and locomotion, and *anxiety-hysteria*, where the condition of dread, anxiety, or fear is the prominent symptom, and is due to some repressed unconscious mental complex. Seventy-seven cases were included in the former group, and 17 in the latter. The third group is the psychasthenics, and these correspond closely with Janet's description of the term. He quotes Hoche, who, on the basis of previous experiences in German wars, places the insane at 2 to the thousand, and thus in an army of $4\frac{1}{2}$ millions there would be about 8,000 to 10,000 insane.

The following conclusions are reached:—War-shock is hysteria occurring in a person free from hereditary or personal psycho-neurotic antecedents, but with a mind more responsive to psychological stimulus than the normal. The wrenching from the customary calling and life, the new discipline, the peculiar and terrible mental strain of modern war conditions acting upon this sensitive mind determine the disease among soldiers. Shell-shock, gas-poisoning, or other physical injuries do not cause the disease. The symptoms are protean—palsies, analgesia, amblyopia, mutism, deafness, affections of the vegetative system such as the soldier's heart, loss of memory, phobias, and obsessions. These result from mental conflict or other mental phenomena, and can be understood without reference to physio-pathology. The psycho-pathology of war-shock is that of the psycho-neuroses. It is thus a variety of hysteria where the one factor (psychic trauma) is overwhelmingly large in relation to the second factor (predisposition). The treatment is hypnotic suggestion, the suggestion being directed to the complex as determined from the psychological examination and general psycho-analytic conclusions. 91·5 per cent. of the above cases of war-shock were cured by this method, and 8·5 per cent. improved. Of cases with previous neuropathic antecedents 62 per cent. were cured, 27·6 per cent. improved, and 10·4 per cent. are unaffected. Cure is rapidly effected in most cases, less than two weeks being required. The usual objections to hypnotic suggestion do not apply to war-shock by reason of the absence of neuropathic antecedents. All methods of treatment, other than psycho-analysis, are based on suggestion, and are more uncertain and less rapid than suggestion under hypnosis. The earlier the patients are treated the better. The majority so cured can return to the Front in three to six months. Cases of "functional" disease should not be discharged from the army until cured.

Much of this book is of a highly controversial nature; many of the facts do not agree with the experience of other workers in similar cases, *e.g.*, the high percentage without a pre-war history, the large number of recoveries, the success of hypnotism, &c., and the number of cases analysed is only 100, but the book gives a good account of these conditions from this special point of view.

Review

of

Neurology and Psychiatry

Original Articles

"STRESS OF CAMPAIGN."

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I.

It is, perhaps, too early to draw definite conclusions or to make any instructive comments as to the relative value of stress and of resistance in regard to the nervous system. It may be that time, and with it a more proper perspective, will suggest amendment in certain directions; but this is inevitable whatever the chosen moment. Later, it is true, the errors we should make might be less egregious; but, on the other hand, it is hoped that the critics will take into account the difficulty of scientific certitude at present inseparable from such a subject.

Even in physics it is not always possible to calculate the relative values of strain and of resistance; in physiology the matter becomes still more complicated; in psychology many of the questions posited can only be answered by means of hypotheses and speculations. This does not mean—as so many infer—that accurate answers are, and will ever be, impossible, but that, at the present time, knowledge is not sufficiently extended to warrant us in doing otherwise. The danger is not in advancing a hypothesis as an explanation; it is in not realising and admitting that it is a hypothesis and nothing more. With such a proviso

as this, it may be allowable to discuss such a subject as the influence of the stress of warfare upon the nervous systems of those participating.

This stress does not, obviously, influence only those who are actually taking part in the campaign; it makes itself felt even among the civilian population. Indeed, with every national upheaval there are associated emotional crises which are the evidence of the effect produced upon the nervous system, especially of those who are unstable. As examples there may be cited such events as the Crusades and the French Revolution. It is to the emotions—primarily at least—that appeal is made. The response is in proportion to the preponderance of emotion over the controlling or inhibitory power of reason. So it is with all popular movements; for we have not yet arrived at a stage of development when intellect is the guiding power. Even where that portion of the brain, the function of which is called "mind," is well developed, it is—being the latest acquired, most complex, least organised—the first to break down when disorder sets in, or when it is impinged upon by the waves of emotional disturbance. The orator, well aware of this, does not make his appeal, therefore, to reason; not, at least, if he is desirous of swaying the multitude. His methods may be empirical, but he knows that they are none the less the most useful. In popular parlance, the appeal is not to the head but to the heart. In the same way during time of war the chief appeal is made to the emotions. It may be made, however, by those who are by no means themselves swayed by emotion or sentiment. They may simulate intense emotional perturbation in order to gain their ends, and adopt a Pecksniffian attitude to gull the credulous. It is not intended here to discuss the rightness or wrongness of war, or whether it is a curse or a benefit; but it may be postulated that there does take place during such a period, psychologically, a preponderance of emotion over reason, and that it is, therefore, a reversal of the process desired by ethical codes. It may be patriotically useful, but it is ethically undesirable.

It is not necessary, however, that these effects of war should be placed in an entirely separate category. It is, says a French observer: "*Une erreur de croire que les émotions des batailles et les commotions des bombardements, si violentes qu'elles aient été, ont créé une espèce ou même une variété de troubles mentaux et*

qu'elles nous obligent à ouvrir en psychiatrie un chapitre nouveau."¹ The tendency is unduly to complicate the subject of the effect of stress upon the nervous system by dealing with the various deteriorative factors as if they produced specific conditions. There appears to be no reason why such an assumption should be made. "The essential pathology of shock is," according to Crile, "identical whatever the cause. . . . When the kinetic system is driven at an overwhelming rate of speed—as by severe physical injury, by intense emotional excitation, by perforation of the intestines, by the pointing of an abscess into new territory, by the sudden onset of an infectious disease, by an overdose of strychnin, by a Marathon race, by a grilling fight, by foreign proteins, by anaphylaxis—the result of these acute overwhelming activations of the kinetic system is clinically designated shock, and according to the course is called traumatic shock, toxic shock, anaphylactic shock, drug shock, &c."² (To which may be added that wide-embracing term, "shell-shock!")

It matters little how energy is withdrawn from the nervous system to an excessive degree; the important thing is that it is exhausted and its function interfered with. If the discharge is regular and along the proper channels, the function is described as normal; if irregular, various terms are used to designate the change. Error creeps in when the words used are understood as meaning something entirely different and not as indicating phases of a process. It is not quite unknown that the meaning of a word may be so altered and its original connotation rendered so vague that eventually it becomes a shibboleth, a fetish, or even more awesome, a "thing-in-itself."

The symptoms or the degrees of response will depend very much more upon the individual who is subjected to the stress than upon the particular stress; and they will vary in the individual relatively to the degree of susceptibility of the different portions of the nervous system. This susceptibility may in turn be due to inherent defect, or may be the result of acquired degenerative processes, such, for example, as those brought about by alcohol and syphilis. The exciting factor is important, but

¹ "Les Troubles Mentaux et la Guerre," by Dr G. Dumas, *Revue de Paris*, 15th July 1916, p. 270.

² "The Origin and Nature of the Emotions," by G. W. Crile, M.D. London, 1915, pp. 219-220.

still more important is the condition of the organism upon which it acts.

"In considering the effects of high explosives, it is absolutely necessary to take into account the state of the nervous system of the individual at the time of the 'shock' caused by the explosive. A neuropotentially sound soldier in this trench warfare may, from the stress of prolonged active service, acquire a neurasthenic condition, and it stands to reason that a soldier who has become neurasthenic through a head injury, or from the acquirement of a disease prior to enlistment, will not stand the strain as well as a neuropotentially sound man. Again, if in a soldier there is an inborn timorous or neurotic disposition, or an inborn germinal or acquired neuropathic or psychopathic taint causing a *locus minoris resistentiæ* in the central nervous system, it necessarily follows that he will be less able to withstand the terrifying effects of shell-fire and the stress of trench warfare."¹ A railway accident or a bursting shell will set up disorganisation in certain nervous systems although no sign of injury may be observed; in others which are more stable no morbid change is apparently brought about, but that there is some alteration we cannot doubt. It may be postulated, too, that there is a certain degree of strain which even the most stable organisation cannot withstand. There must be a breaking-point. The stress, again, may be applied suddenly or it may be spread over a long period; but it will be difficult in many cases to deduce whether the cause has acted rapidly or slowly, so similar are the effects.

II.

It is becoming increasingly difficult to differentiate, with the complacency which characterises many writers, between the various stimuli in relation to the human economy. No doubt exists in their minds that physical and psychical stimuli or shocks may be put into entirely separate categories. Yet we begin more and more to question the validity of this assumption. Is there really a dividing line here, any more than there is in the animal kingdom, between man and the lower animals? Or is it not a

¹ "The Effects of High Explosives upon the Central Nervous System," by F. W. Mott (Lettsomian Lectures), *Lancet*, 12th February 1916, p. 331.

question of degree rather than of kind? "In their quality and in their phenomena psychic shock and traumatic shock are the same."¹ When we consider the amoeba on the one hand and man on the other, we find it difficult to realise their kinship and their common ancestry; but, as link after link is added to the chain, the difficulty of the conception disappears. So with mental processes; from diffuse irritability up to the thought there is apparently no definite dividing line. "From the amoeba, on through all the stages of animal existence, every action is but a response to an adequate stimulus."²

For purposes of convenience arbitrary divisions have been made; and now we are apt to consider the efflorescence as something totally unconnected with the plant, and still less with the roots!

Research is tending to demonstrate that even emotional shocks are accompanied by changes in the brain-cells; or, rather, the emotions are really *commotions* in the cells. They are responses to stimuli. The difficulty in realising this lies in the fact that the response is not proportioned to the amount of the stimulus, but to its character. "The part played by the receptor in the reflex-arc is in the main what, from other evidence, it is inferred to be in the case of the receptors as *sense-organs*; namely, a mechanism more or less attuned to respond specially to a certain one or ones of the agencies that act as stimuli to the body. . . . The main function of the receptor is therefore *to lower the threshold of excitability of the arc for one kind of stimulus, and to heighten it for others.*"³ Consciousness, resulting from the functioning of lately developed cells, is brought about when the discharge from other areas overflows them, or when it passes along nerve-channels gradually formed by recurring stimuli from childhood onwards. "Consciousness itself is a reaction to environment, and its intensity must vary with the state of the brain and with its environmental stimuli."⁴ Some stimuli are delayed, and this process is designated psychologically "self-control." Others sweep across, disregarding the controlling areas, and spontaneous action takes place. These areas may be conscious of what is taking place, but are unable to check

¹ Crile, "Origin of Emotions," p. 76.

² *Ibid.*, p. 149.

³ "The Integrative Action of the Nervous System." C. S. Sherrington, London, 1906, p. 12.

⁴ Crile, "Origin of Emotions," p. 140.

it. In the old days this was explained by saying that people were possessed by devils. They saw and knew the better way, but followed the worse.

Fear and anger are subjective states—nervous responses to certain stimuli. The stimuli in themselves are not fear- or anger-producing, but only become so when they impinge upon sense-organs, and are thence conveyed to the brain, which, depending on education and on inherent or hereditary characteristics, responds in a certain way. For example, the doings of the Germans in Belgium produce in us a feeling of anger or of disgust; but in many of the German people they give rise to satisfaction or even to joy. Fear is the subjective aspect of, or nervous response to, noxious stimuli. It is, however, dependent not only upon these stimuli, but also upon the perception or realisation of them. This being so, it is evident that courage is also a relative term. What is apparently a courageous act may be simply a result of lack of appreciation of the dangers. When a cow charges a railway train we do not feel inclined to commend its courage in so doing!¹ On the other hand, where there is realisation of danger, and yet action in the face of it, courage is admitted; and in such a case it may be said that the greater the fear the greater the courage. Such action is dependent—it may be again stated—upon inhibition by “higher” cells of the impulses which would otherwise flow over into the motor areas which ordinarily respond to such stimuli, and upon the passage of those impulses which result in courageous conduct. This capability or directing power is itself the result of both nature and nurture. According to Crile: “When our progenitors came in contact with any exciting element in their environment, action ensued then and there. There was much action—little restraint or emotion. Civilised man is really in auto-captivity. He is subjected to innumerable stimulations, but custom and convention frequently prevent physical action. When the stimulations are sufficiently strong, but no action ensues, the reaction constitutes an emotion. A phylogenetic fight is anger; a phylogenetic flight is fear; a phylogenetic copulation is sexual love.”²

¹ “For as to fear, in all encounters, is the mark of a heavy and cowardly heart . . . so not to fear when the case is evidently dreadful is a sign of want or smallness of judgment.”—Rabelais, “Pantagruel,” Bk. iv., ch. 23.

² Crile, “Origin of Emotions,” p. 76.

Again, where the realisation of danger is intense, and yet where self-control is sufficient not only to inhibit fear but to allow of courageous action, the strain is greater and the possibility of exhaustion increased. "There is evidence that the effect of the stimulus of fear upon the body when unaccompanied by physical activity is more injurious than is an actual physical contest which results in fatigue without gross physical injury. It is probable that the various energising substances needed in physical combat, such as the secretions of the thyroid, the adrenals, &c., may cause physical injury to the body when they are not consumed by action."¹

This difference is well illustrated by the story of the old campaigner and the youth new to battle-fields. The youth, observing that the veteran trembled when the shells began to whizz past, remarked, "I believe that you are afraid!" "Yes," replied the other, "and if you were half as afraid as I am you would run away!"

"The psychology of fear is a strange thing. It is, perhaps, paradoxical, but I venture to think that without fear there can be no bravery—bravery, that is, in the true sense of the word. There are, I believe, some men who are without fear—literally and absolutely fearless. Such a condition of mind may be induced by sincere fatalism, but I rather think that in the majority of cases it is due to a peculiar and fortunate twist of the brain. . . . It may well be that, were the wonderful, soul-stirring heroism of some V.C. to be weighed in the balance of mind and soul rather than in the balance of deed, he would be found less worthy to hold that coveted ribbon than a man whose sole contribution to fame was that he didn't run away."²

Such strain as this implies great tension in the nervous system, and it is likely if prolonged to lead to exhaustion. When the stage of exhaustion is reached it is difficult to say in what way it will show itself. It may have the aspect of fatigue or neurasthenia. The term "neurasthenia" is, however, rather a vague one. It is applied to conditions which, seen in their entirety, would have to be classified otherwise. There may be merely the asthenia, the general nervous weakness and loss of tone. On the other hand, cases which have been labelled as

¹ Crile, "Origin of Emotions," p. 64.

² "The Lieutenant," by "Sapper." London, 1916, pp. 123-125.

neurasthenia have proved to be—early or late—phases of such conditions as mania, melancholia, or even early cases of general paralysis of the insane.¹ There is diminished power of effort; the least exertion is tiresome; there is a feeling of inability to perform even simple duties. Prolonged rest may not suffice to allow of recuperation; and months may pass before this *tedium vitæ* disappears. In other cases, there is as the most prominent symptom what may be described in the popular phrase as “loss of nerve.” A man ordinarily courageous becomes timid and apprehensive: the thought of dangers which he would ordinarily face unflinchingly, renders him incapable of action. Added to this there is, at times, a condition in which he is afraid of being afraid; he fears that he may fail at the critical moment to do what is required of him.²

When the condition is more pronounced there is still more obvious loss of control. The patient is tremulous, and his aspect is one of terror and apprehension. The tremors may be of such a degree that he cannot stand or walk; even when he lies in bed there are constant twitching movements. In some cases the patient appears as if he were ducking his head at the approach of a shell. Occasionally the condition resembles chorea. Added to this there are, quite frequently, terrifying dreams. These may take the form of recapitulation of events which have happened whilst the patient was in the trenches—of shells bursting, of gas attacks, of mines exploding. Some of those who suffer in this way start up suddenly in the night, shouting to those about them to take cover. Sleep is disturbed; there is restlessness and, where the dreams are very terrifying, there may even be disinclination to go to sleep because of dread of these nightmares. This condition is vividly described by a German novelist: “He continually dreamed of the roar of cannon, and awake he saw blood and corpses, corpses and blood. Everything he looked at seemed tinged with that terrible crimson. He was scarcely able to swallow a mouthful, because his food seemed soaked in blood. For the slightest cause or none, he would weep like a little child. These terrible fits of weeping

¹ See article on “Neurasthenia” in the “Dictionary of Psychological Medicine.”

² An interesting case is given by Prof. G. Elliot Smith, “Shock and the Soldier,” *Lancet*, 15th April 1916, p. 917.

would come on at any moment, exhausting him body and mind. It seemed as if the tears of the women and children of Rosez, whose homes he had ordered to be set on fire, had been collected that they might flow from his own eyes."¹

Mutism is not uncommon. There may be no signs of injury, nor even the other symptoms associated with "shell-shock," but the patient is unable to speak. This may last for days or weeks, and it may pass off suddenly. Many instances have been recorded, and certain of them, because of their dramatic nature, have attracted attention and been noted in the newspapers from time to time. One patient falls into the water and immediately recovers his power of speech; another sees an accident imminent and shouts out a warning; a third, listening to a familiar chorus, joins in the singing. In another case, a young soldier who had been in the retreat from Mons and much subsequent fighting, the excitement of being transferred by train and motor from one hospital to another appeared to have a stimulating effect. On arrival he spoke in a whisper, and the next morning speech was normal and remained so. In one instance mutism resulted where a shell fell near but did not explode. Speech returned some weeks later. In some cases there is hesitant speech or stammering before complete recovery takes place; and some of these have "stammered, stuttered, or suffered with a hesitant speech at some time in their life prior to the shock."² There may have been exposure to definite shock preceding the onset of the mutism,—from high explosives or from being buried by debris; and, at times, wounds from bullets or from fragments of shell may also precede the speech defect. On the other hand, there may be no history of definite trauma. The speech-defect may be part of a general exhaustive process; and it may pass off as improvement in health takes place, though, in some instances, it persists even after this. Again, the mutism may be apparent—that is, there may be no real interference with the speech-mechanism, but the condition may be associated with the stuporose state. In these patients there may be prolonged mutism; or, from time to time, they may utter a few words or sentences quite clearly and distinctly. Where the trouble is of a "functional" nature, the resistance or inhibition may be overcome by means

¹ "Love's Inferno," by Edward Stilgebauer. London, 1916, p. 214.

² Mott, "Lettsomian Lectures," 1916.

of some local electrical stimulation applied to the pharynx or larynx. In these cases the loss of the power of speech is apparently due to a transient interruption in the nervous speech-mechanism, or it may be the result of interference with other areas and a secondary inhibition of speech.

Deafness also occurs in certain cases. It may be due to some transient nervous disturbance or to a permanent change in the ear or in the brain. The tympanum may be ruptured on both sides or on one alone. In the transient conditions it may pass off rapidly. In one instance where there was also mutism, and where the mental state was one of stupor, the improvement was rapid in all directions; the patient was able to hear and to talk in the morning although he could not do so the night before.

The sense of hearing may be exaggerated—there may be hyperacusis. Even slight sounds may give rise to acute discomfort; and where, added to this, there is increased motor irritability, the patient may start violently at an apparently quite inadequate stimulus. There may be auditory hallucinations. The patients hear buzzing, booming, or whizzing noises; in addition to these there may be "voices." In the types of case already mentioned the patient is almost invariably aware of the subjective nature of these. There are others, however, where this power of realisation is lost, and where, therefore, definite mental disorder is brought about.

The superficial and the deep reflexes may not show any alteration worthy of note. Generally, however, the tendency is towards exaggeration. Even in the hemiplegias and paraplegias, which are not due to gross interference with the central nervous system, the reflexes are of the "functional" type; although there may be, for example, a Babinski sign in certain cases which alters to the plantar type as improvement takes place, illustrating the difficulty of dogmatising as to whether the condition is "functional" or "organic." Rombergism is not unusual; as a rule it is, apparently, a part of the general tremulousness and loss of muscular tone which are such prominent symptoms. The pupillary reaction is frequently brisk, and the pupils may be dilated.

The ordinary manifestations of hysteria, such as monoplegias, paraplegias, and hemiplegias, with or without sensory disorders and muscular contractures, affections of the special senses, &c., are

not infrequent, and are certainly met with more often than at ordinary times.¹ The nervous disorders associated with stress of campaign ("shell-shock," &c.) have received much attention, and many interesting papers have been written. It is hardly necessary, therefore, to repeat what has been said therein, and they are only dealt with incidentally here. In addition to Major Mott's "Lettsomian Lectures," there may be mentioned, in particular, Lieut.-Col. C. S. Myers' "Contributions to the Study of Shell-Shock," *Lancet*, 13th February 1915, 8th January, 18th March, and 9th September 1916; "Some Neuroses of the War," by Lieut.-Col. J. Michell Clarke, *Bristol Med. Chi. Journ.*, July 1916; "Shock and the Soldier," by Prof. G. Elliot Smith, *Lancet*, 15th and 22nd April 1916.

III.—MENTAL DISORDERS.

As has already been stated, no clear line of demarcation can be drawn between these disorders and those existing in other parts of the nervous system. They merge one into the other indefinitely. So classification is difficult. Yet where the mental symptoms are predominant, it is convenient to describe such cases as suffering from mental disorder; and a further classification may be made by those who are desirous of distinguishing in order to divide, by denominating the more disordered as cases of insanity. The strain may not fall primarily upon these areas of the brain. Other parts of the economy may have had their energy sapped and may have broken down. So it comes about that patients sent into hospital with symptoms pointing to disordered conditions other than nervous ones may eventually migrate to the mental department. In most of these cases, however, the point of least resistance is in the nervous system, and it is there that the breakdown occurs. Here again, however, the disorder is located in certain parts; or, at least, it is in them that disorder is predominant. It may be transient or it may be the beginning of a degenerative process. In other cases there is aggravation of disorder already present, or defective development may be rendered more obvious. As with other parts of the nervous system, it is

¹ See especially "Some Neuroses of the War," by Lieut.-Col. Michell Clarke, *Bristol Med. Chi. Journ.*, July 1916.

difficult to say that there is any specific disorder associated with stress of campaign. There does not, therefore, appear to be any pressing necessity to coin neologisms to suit the occasion. This is not to say that there are no difficulties in regard to classification; if that were so it would imply that in insanity it has been easy to allocate cases to their proper classes—which is notoriously not so. When one considers how comparatively limited is our knowledge as to normal mental processes, it is hardly to be expected that abnormal psychology should have arrived at a stage where exactitude of classification is possible. Until more exact pathological information is available, classification must be provisional.

Perhaps the most characteristic mental state observed among those exposed to stress of campaign is one of confusion. This varies in different cases—or in the same case at different times—from a condition where the mental processes are apparently quite chaotic, to others in which it resembled more a condition of day-dreaming. In the more pronounced examples there is incoherence of speech, inability to understand questions, defective memory, disorientation; the patient is unable to concentrate his attention or occupy himself; he may wander restlessly about or sit staring vacantly. Some of these patients are extremely emotional—weep when spoken to, are melancholic, childish, and plaintive; and the occurrence of depression along with confusion is more frequent than is usually to be observed—indeed the ordinary melancholic patient is comparatively little impaired as to memory and orientation. For the most part these symptoms clear up in the course of a few weeks; the general health—which has been in most cases poor—improves; and a good recovery takes place. This confusional state may, however, be an early stage of some progressive disorder, such as dementia præcox or general paralysis of the insane. It is not possible to draw any hard and fast line between these cases and others in which there is definite stupor. In the latter, instead of difficulty of cerebration and more or less spasmodic mental action, there is apparently a total suspension of intellectual activity. In all of them there is interference with memory; which may be anterograde or retrograde. In the confusional cases the defect may be so pronounced that the patient is unable to recollect even his name, and he has no remembrance of where he has been nor what has happened to him. In spite of this there may, however, have been fixation of certain memories; and in the course of time

there is a revival of impressions which had apparently left no lasting effect.

The confusional state may be brought on rapidly by some such drastic disorganisation of cell structure as that resulting from a shell explosion, or it may be induced by long-continued exhaustion. Again, as Mott and others have pointed out, the absorption of CO gas may have exerted a prejudicial action through the blood stream on the cerebral tissues. It is conceivable, therefore, that the defect is due either to a diaschizis—a retraction of dendritic processes—or to cellular deterioration. It is obviously impossible to say how far these changes have gone. In stupor, for example, there is to the casual observer an almost entire annihilation of mental processes which may be due to cellular deterioration, and is, therefore, a condition of dementia. Yet, as is well known, there is very frequently recovery from the striking state of hebétude which characterises stupor; and so it is with the conditions of confusion and stupor now under discussion. Until, however, recovery takes place it is not possible to state how far cellular deterioration has progressed. Even then, with our rough and ready methods of gauging mental capacity, and with no experience of the former capabilities of our patients, we cannot say in a particular case whether or not recovery has been quite complete. Crile has shown that when stimulation has been carried to a harmful extent some of the cells remain permanently deteriorated. Consequently we cannot be sure that the individual is just as fit mentally as he was before being subjected to the strain. In calculating the disability it will, too, be necessary to take into consideration whether or not the patient's occupation is manual or mental. Deterioration which would be of little practical account to a labourer may make a grave difference to him whose livelihood and chances of advancement are dependent upon his intellectual ability.

Stupor may be of the anergic or of the resistive type. In both there is, in many instances—and sometimes for prolonged periods—entire refusal of food, and a consequent necessity of resort to tube-feeding. The habits may be faulty. The patient does not speak, or from time to time he may give utterance to a few words or sentences and then relapse again for weeks or months into a dull, apathetic, taciturn, and apparently mindless state. It will be seen that these cases, therefore, conform generally

to the ordinary clinical types. The stupor may come on months after the acute strain—explosion, prolonged stress, wound, or whatever it may have been—and in this it differs from the confusional condition which usually follows more or less directly upon the causative factors.

It will probably be found in these cases that those who most readily succumb to the effects of strain and stress are hereditarily unstable. The potentiality is present here as in the neurasthenic. Histories, as far as they can be ascertained, bear this out. On the other hand, it is necessary to admit that in some instances no such evidence is present—or ascertainable; and the stress has, therefore, in these latter cases been too great for stable nervous systems.

MANIC-DEPRESSIVE INSANITY.

Many cases of mania have occurred; and in these it has frequently been difficult for a time to decide whether or not this has been a phase of some disorder such as primary dementia, epileptic insanity, alcoholism, or even general paralysis of the insane. Theoretically it should, of course, be a simple enough matter to discriminate; but, unfortunately, it is not by any means invariably so in practice. Indeed a considerable time may have to elapse before a decision can be arrived at with any degree of certitude. The difficulty is frequently increased by the fact that the intense excitement renders accurate physical examination impossible; but even when this can be carried out it does not solve the question as to whether one is dealing with, for example, a case of primary dementia, or with a phase of a commencing manic-depressive state. In regard to the latter condition it seems that here, as in ordinary practice, we have not yet arrived at a stage when we can place all manias and melancholias—that is to say, those which are not phases of other conditions such as those mentioned above—in the category of manic-depressive insanity. It seems more convenient to adhere to a classification which allows of the inclusion of mania and melancholia as conditions distinct—at all events hypothetically—from manic-depressive insanity, reserving the latter term for those cases where there are cyclical recurrences.

The mania is, as ordinarily, of all degrees of severity, from

simple excitement without hallucinations or delusion or any pronounced physical signs, to those in which the patients are noisy, restless, sleepless, resistive, and incoherent. In some instances the acute symptoms have developed whilst the patients have been actually undergoing the stress of active service; in others the attack has not resulted until perhaps weeks or months after they have returned from abroad. In the latter cases it is then difficult to say how far the condition has been aggravated by the strain undergone whilst they have been abroad: indeed in almost every instance it is not easy to decide as to how far the attack has been precipitated by exposure to stress.

In one of the most acute cases of mania the symptoms appeared about a fortnight after amputation. The patient had been wounded in France. Amputation was carried out by the guillotine method through the right leg and through the left thigh, the day after he received his wound. When he was admitted the stumps were suppurating. He was wildly maniacal—shouting, throwing himself about, and removing his dressings whenever an opportunity occurred. He was quite incoherent, and, of course, entirely without any appreciation of what he was doing. Sleep was only obtained by means of hypnotics. It became necessary to pass a sheet round his elbows to prevent him from interfering with his dressings. This restraint was not resented in the slightest; indeed he was cheerful—if such an acutely excited state can be considered as cheerfulness. By day and night, except when rest was procured by means of hyoscine, sulphonal, or paraldehyde, he would shout and sing, waving his stumps in the air. His general health on admission was poor. The excitement lasted for two months; then, in spite of the strain of wounds and mental trouble, he began to improve. Three months from the time of admission he was transferred to the surgical side. He remained well mentally, and had no relapse even when re-amputation was done. He was eventually discharged, completely recovered, eight months after admission.

A number of cases might be quoted in which maniacal symptoms occurred, in many respects similar to this one, of varying degrees of severity, and with wounds of different parts of the body. It may be a mere coincidence but the majority of the cases which have developed maniacal symptoms have had wounds or injuries of the lower limbs. In several cases

the maniacal symptoms have been associated with trench-foot, or with frost-bite involving the toes.

Recovery has been the rule in these cases as it is in ordinary practice—that is to say, when the condition is one of mania which is not a phase of some other mental disorder.

One case of delirious mania associated with a severe wound of the shoulder succumbed to the exhaustion arising from this and from broncho-pneumonia. In the early stage of this patient's attack it was necessary to resort to artificial feeding; and in several other cases of mania the same method had to be adopted.

Melancholia has been of frequent occurrence. It, too, is observed in all degrees, from mild depression, without any apparent delusions or hallucinations, to acute melancholia, in which the latter symptoms may be very noticeable. Quite a number of the more acute cases have been resistive and agitated; and not infrequently it has been necessary to resort to tube-feeding for prolonged periods. So closely do these cases conform to the usual clinical types that it is not necessary to do more than merely refer to them. Here also the prognosis is quite good. The majority recover completely—even those cases where, as a result of prolonged excitement, agitation, or refusal of food, the general health has become very greatly impaired.

The melancholic symptoms supervened in a number of cases in association with wounds. How far the wound was the exciting factor it is not possible to say; it may, however, have been the final aggravation to a brain already wearied by stress.

Although it is not possible—in view of the comparatively short time the patients are under observation and in the absence of reliable histories—to say that the maniacal or the melancholic attacks are phases of manic-depressive insanity, there have been under observation a few cases in which the alternating conditions have been noticeably present, and in which the whole cycle has occupied but a short time. Although there was apparently previous instability, there could be obtained no history of rapidly recurring manic-depressive phases. In the cases under consideration the fluctuations occurred over a period of from nine months to a year; thereafter stability appeared once more to have been regained—though it is obviously too early to state whether it will be enduring. Whatever the etiological factor may be in this form of insanity, experience only too sadly shows that, the periodicity

being established, the tendency is for it to persist to the end of life. There may, of course, be a toxic factor; but often enough—and especially in the milder forms—there appears to be an accentuation of the usual mental variability rather than a disease brought about by, for example, a microbial infection. On the other hand, it is just this periodicity which characterises certain organismal infections. So, until knowledge becomes more precise, the settlement of the question must naturally remain in abeyance.

In one of these cases (Rifleman P. K., æt. 27) there was a history of an attack seven years previously; but from that time he had apparently had no recurrence. He went to France in November 1914 and remained until January 1916; during that time he saw much fighting and was frequently under fire. As far as can be ascertained he kept well mentally throughout all this period of stress. In April 1916 he had an attack—the nature of which is not specified—from which he recovered. He was sent on leave; returned to duty; but shortly thereafter broke down and became maniacal. This was followed by a period of depression. From the end of June 1916 until the middle of December 1916 he had six maniacal-depressive attacks. These were of diminishing intensity both as regards the mania and the depression. Since December 1916, until the present time, he has remained well.¹ It is interesting to note that in April 1916, prior to his breakdown, he cut his hand with a tin: the wound became septic, and he had to go to hospital with it. This toxic absorption may possibly have been the exciting factor.

Rifleman E. (æt. 21) was in France from July 1915 till January 1916. He was in the trenches and under fire, but was not wounded or otherwise injured. In January he had a sharp attack of mania—he was restless, incoherent, confused, sleepless. From this time until May of the same year he had five maniacal-depressive attacks. In the excited stage he shouted, sang, was restless, and slept very little. From May till September there was no recurrence of any acute symptoms; and, as he then seemed more stable, he was discharged to his home.

In neither of these cases was there any history of alcoholic excess nor of venereal disease. Rifleman K. was unstable—as is shown by the history of a previous attack; while the father of

¹ June 1917.

Rifleman E. is stated to have died insane. It is interesting to note that K. did not break down mentally until some three months had elapsed after he left the trenches, and in his case there was septic absorption as an additional factor. It would, however, be unfair to allow no weight to the possible influence of the period of active service in bringing about his breakdown; and the same may be said of other patients who, after prolonged exposure to the stress of fighting, are sent to the base or to England for some condition other than mental trouble, and who have an attack of mental disorder weeks or months later.

These two cases have been adverted to in order to call attention to the possible effects of the stress of actual warfare. But, on the other hand, the same train of symptoms may be brought about in others who have not been so exposed, as, for example, in civil life. The point of interest is whether in the cases described the ordinary stresses of civil life would have sufficed so to accentuate their nervous instability. The question cannot be answered dogmatically; the odds are, however, in favour of the hypothesis that the stress was certainly the most potent factor.

In a third case (Sgt. P., æt. 31) the question is still more difficult. He went to France at the end of March 1916. He was not under fire nor in the trenches. On the 10th of May 1916 he is reported to have been "sleeping badly for a week"; he had loss of memory, he acted strangely, and he worried about spies. He was suspected to be suffering from general paralysis of the insane—but this was not confirmed after prolonged observation. He was sent home in June 1916, and from the beginning of that month until the end of November 1916 he had six recurrences of the manic-depressive condition. The phases of excitement were characterised by symptoms which were at first suggestive of dementia præcox, such as grimacing, posturing, laughing foolishly, talking in a foolish, silly, irrelevant way. The symptoms generally became less pronounced in the succeeding attacks; and from November until the time of his discharge from hospital (April 1917) he kept well. No definite history of any previous mental trouble could be elicited, nor of alcoholic excess.

It is not, of course, possible to say whether or not lasting stability has been regained in these cases. Time alone will show. It may be that return to civil life and no further exposure to

the ordeal of active service will allow these faulty mechanisms to carry on such work as they are capable of. Nevertheless, in view of the acuteness of the attacks, it is allowable to feel some anxiety as to their future.

Suicide.—One of the most striking features of the cases of mental disorder has been the frequency with which suicidal attempts have occurred. It is not possible yet to give any reliable statistics as to these attempts; but that they have been more numerous than in ordinary experience seems to be certain. The method most usually adopted has been that of cutting the throat; and the number of attempts in this way is far in excess of all others. For the most part the razor has been the most favoured implement, though knives and pieces of glass have also been employed. In one case where the throat had been cut by means of a razor some months previously, a further attempt was made by means of a sharpened piece of tin. It is almost incredible how badly the throat may be gashed and yet a fatal result not be brought about. Indeed, it is the exception. When one of these attempts has been made by means of cutting the throat there is not usually another endeavour in the same way. This is not by any means so where strangulation is the method chosen; many and determined efforts may be made, and such cases are, therefore, an even greater source of anxiety than is the usual suicidal patient.

In one case the patient, an Australian, had been wounded in the head with a resulting right hemiplegia. He became acutely depressed and cut his throat badly with a razor held in his left hand—without, however, a fatal result. In another case the incision commenced in the middle line of back of the neck and finished under the lobe of the ear, passing fairly deeply into the sterno-mastoid muscle. In a third the incision was perpendicular, just missing the carotid artery.

Precipitation and poison have also—but less often—been tried. Perhaps one of the most remarkable things is that shooting has very seldom been the method chosen.

Suicide is usually attempted in a state of depression, though it may be during a maniacal phase. This may be due to a condition such as melancholia; but in a large proportion of the cases seen the attempts have been associated either with acute alcoholism or with the intense depression which so frequently

follows such excess. It is apparently but seldom that suicide is resorted to as a method of evading military duties, though such cases do occur. Many of the attempts have, however, been made by soldiers who have been through long and strenuous periods without any apparent ill effect, and who have not any immediate prospect of return to the battle-field. This is in accordance with ordinary experience. It is not those who have a definite ordeal to face who act in this way. Their energies are directed in other channels: whereas in others there is no definite outlet, and there is in consequence emotional stress with a possible misdirection of energy.

It is no more easy to decide upon the basal factors in these cases than it is in those occurring in ordinary times. It is a puzzling symptom which may be dependent on different causes. It appears, however, to be associated in many cases with an exhausted condition of the nervous system. It is an interesting fact that suicide is more common in the hot weather—or at the onset of the hot season—when more demand is made upon the nervous system, and when the period of rest and recuperation is not as satisfactory as during the cold period. As the summer progresses and as the body accommodates itself to the altered conditions there is, almost invariably, a decline in the suicide rate.

Self-inflicted injuries to the hands, feet, knee, &c., occur during war-time, and the present campaign has been no exception to this rule. The cases appear to have been very few when one considers the huge number of men involved. Even among those who have injured themselves with a view to evading duty, quite a large proportion have been rendered so unstable by the altered conditions of life and by other factors that they could by no means be considered responsible for their acts. The potentiality for accommodation to environment varies so greatly in individuals that it has not been necessary in some cases that they should be exposed to the drastic alterations entailed by active service. So it has been that during training some have become so—biologically—out of sympathy with their surroundings that they have attempted the most drastic measures in order to escape from them. It seems to be that in war the fit endeavour to eliminate one another; while a certain number of the unfit—some congenitally so and others deteriorated by stress—direct their energies upon self-destruction.

DEMENTIA PRÆCOX (PRIMARY DEMENTIA).

This is a condition which—even in ordinary times—gives rise to much discussion. Patients exhibit symptoms which are so varied that it is difficult to know whether a particular case can rightly be included in one of the categories of this disease. Where there is still such a flux of opinion much is left to be decided in accordance with individual predilection. Yet there can be no doubt that, however much we may criticise Kraepelin's description or his claim to priority, he has done a very great deal towards elucidating a difficult subject. Great as the difficulties are in ordinary times in coming to a satisfactory diagnosis, they are still further increased at present. Mention has been made of the confusional state so common in the patients exposed to severe stress; and it has been noted that, underlying it or following on it, may be this condition of primary dementia. On the other hand, among these confusional cases are some which appear at first to be typical cases of dementia præcox, but which clear up completely and would falsify one's predictions were a too decided opinion emitted during the early stages.¹ A further difficulty lies in the fact that, even in dementia præcox, remissions of symptoms may occur; so that here—as in general paralysis of the insane—too much stress ought not to be laid on an apparent recovery. In addition to the confusional state there may be also a preliminary period of excitement. The excitement and restlessness recur on many occasions with only short intervals of quiescence, very similar in many respects to manic-depressive insanity; but in contradistinction to it there is a steady, often fairly rapid, mental deterioration. In other cases the preliminary stage is one of depression, it may be with hypochondriacal delusions. It seems a reasonable hypothesis that, in these instances, there is, in addition to the general deterioration, a more selective action of the deteriorative factor, so that at first the true underlying condition is masked by the more prominent mental symptoms.

It seems probable that no one factor can be assigned as the

¹ Major Hotchkis draws attention to this difficulty in regard to the patients treated at the Dykebar Military Hospital. "There are," he states, "a number of cases recently admitted who were classed provisionally under manic-depressive insanity, but who will probably ultimately prove to be cases of dementia præcox."—"A War Hospital for Mental Invalids," *Journ. of Ment. Science*, April 1917, p. 246.

causative agent in such a condition as dementia præcox. What appears more likely is that there is hereditary instability of the nervous system which renders it prone to deteriorate rapidly either under the normal stress of life or, *a fortiori*, when it is subjected to any unusual strain, a strain which would not upset the equilibrium of, let us say, the average individual. The likelihood of this being so is greater in that, in probably the great majority of cases of dementia præcox, a history of mental disorder in the patient's family is ascertainable. This is, too, the more borne out by the fact that a certain number of cases have developed definite dementia præcox subsequent to head injury; but obviously there must be some other factor than the injury to be taken into account. The same may be said of general paralysis of the insane; for, of the many who suffer from syphilis, only a small proportion afterwards are afflicted in that particular way.

The trauma acts apparently like a crystal dropped into a saturated solution, and becomes a focus wherefrom degeneration spreads. It is not, however, impossible that even the healthy brain may degenerate if the trauma is of a particular kind, and is associated with, for example, insufficiency of secretion of one of the ductless glands.

In one case the trauma took the form of compound fracture of the skull over the right Rolandic area, with opening of the dura mater and laceration of the brain. After the injury the patient was unconscious for a short time, but thereafter he was able to walk out of the trench with the aid of two of his comrades. On the same day the wound was opened up and depressed pieces of bone were removed from the brain substance, which bled very freely. The wound healed, but hemiplegic symptoms supervened. These passed off gradually, and power was regained in the arm and in the leg. Some facial paresis, however, remained. A little later a mental change was noticed; he became restless and excited, impulsive, and, at times, difficult to control. The excitement passed; but already mental deterioration had taken place. Later he exhibited tricks and mannerisms; his memory became more and more impaired; and he steadily passed into a childish, mentally enfeebled state.

Although in this case no definite history of mental trouble in the family could be obtained, there was evidence of nervous instability, especially maternal.

In other cases progressive mental deterioration has been noted following head injuries; and in some of these, although the symptoms were not so clearly defined and so typical as in the one described, there was much that gave rise to the impression that some very similar change was taking place. This possibly may be the case in organic dementias following traumata.

GENERAL PARALYSIS OF THE INSANE.

The most interesting feature of these cases is that apparently the average age at which the symptoms are noticed has been lowered. It is not possible to speak dogmatically as to this, but it is a not unexpected result. It is obvious that some factor is necessary, other than the mere organismal infection, before the central nervous system is involved. As in cerebro-spinal fever, there must be some lowering of resistance before the further infection takes place, or, after the organisms have penetrated to the central nervous system, before they grow and multiply sufficiently to give rise to definite symptoms. It has already been shown that the various stresses associated with active service tend, if prolonged, to bring about deterioration of the nervous system. It is not, therefore, illogical to infer that in an economy so denuded of its resisting power certain organisms may thrive and prosper; whereas, if the body had been able to conserve its energy, their virulence might have been checked and their propagation deferred.

It may be that the cases have come earlier under observation than they would have in normal times, when the duties which such patients would have to perform could be carried on in a more automatic manner. It is indeed remarkable how, in the ordinary way, the symptoms of mental disorders pass unnoticed; and a comparatively advanced stage of, for example, such a condition as general paralysis of the insane may have been arrived at before the friends and relatives of the patient begin to suspect that all is not well. This is less likely when the men are being passed regularly in review by the doctor. Even this is not apparently sufficient to account for the discrepancy; and it seems more likely that the disease has been aggravated by the additional stress.

The more one sees of cases, the more impressed does one become by the fact that the clinical diagnosis of general paralysis

of the insane is a matter of extreme difficulty. This may seem an amazing statement to those who are accustomed to think that it is one of the diseases most easily diagnosed. Nevertheless it is a fact. It does not, of course, follow that all the cases are difficult of diagnosis. Many are as simple as even a text-book description could lead us to infer. Altered reflexes, tremors, Rombergism, unequal pupils, slurring speech, grandiose ideas, excitement, loquacity, &c., are all there for our guidance, and diagnosis is a straightforward matter. Notwithstanding this there are other cases where suspicion is aroused, but where the physical signs are not sufficiently satisfactory to enable a definite statement to be made as to the underlying condition. Recourse is, therefore, necessary to the pathological findings; and if the Wassermann reaction in the cerebro-spinal fluid is positive—while at the same time there is an increased lymphocyte count—in a case which clinically suggests general paralysis of the insane, the diagnosis is practically confirmed. It is not, therefore, surprising that cases of general paralysis of the insane have been admitted with a diagnosis of neurasthenia, “shell-shock,” and so on; and in these cases above all the agnostic, non-committal “N.Y.D.”¹ is a very present help! Indeed it would be unfair to deny that these diagnoses are actually wrong. For example, some of these cases have been exposed to the concussion of bursting shells or of mine explosions, and have exhibited for a time indubitable evidence of the shock to which they have been subjected. If it be true that the underlying trouble can be aggravated by such stresses, it is reasonable to suppose that these other conditions may be superinduced before the symptoms of general paralysis make their appearance. Where this has occurred the diagnosis has certainly been rendered more difficult. Exaggerated tendon reflexes and general tremulousness—involving the face and tongue—are so common in those admitted that little help can be derived from their presence; and even the most meticulous fail at times to criticise the condition of the pupils in cases which have proved later to be general paralytics.

The cases definitely diagnosed have conformed to the ordinary types—exalted, melancholic, or progressive mental enfeeblement without excitement or depression.

¹ “Not yet diagnosed.”

It is almost needless to add that alcoholic excess is another factor which complicates the matter of diagnosis in these cases.

ALCOHOLISM.

The effect of alcoholic excess has already been adverted to in regard to its influence in bringing about suicidal attempts. There appears to be no doubt as to its potency in this direction, and recent experience tends to confirm the opinion that suicide is one of the symptoms to be anticipated in many cases of alcoholic excess. The number of cases among the soldier-patients in which this symptom has occurred is, however, disproportionately great in comparison with those observed in ordinary times. Cut throat has been by far the most common method in this condition as in suicidal attempts in general.¹ Many of these attempts were made during an acutely confusional stage, and, later, there was no recollection—or apparently none—of what had taken place. In others it was associated, as has been already noted, with the intense depression which alcoholic excess produces in certain individuals.

Cases of delirium tremens have occurred, but these have been among soldiers home on leave or among those who have been invalided home for other reasons and have gone on a drinking bout when opportunity arose. It may be that their exposure to stress had rendered them more susceptible to the effects of alcohol: and this is, indeed, a statement quite frequently made by the patients themselves. Especially is this so where a definite head injury has been received: and it does not seem unreasonable to infer that others without obvious injury may have suffered in less degree reduction in their tolerance of alcohol.

The excitement associated with alcoholic excess does not, in the ordinary way, invariably reach the pitch of delirium tremens. There are stages between that and the ordinary exaltation produced by alcohol.

"Between these two classes [delirium tremens and chronic delusional] were those who showed various symptoms, as confusion, depression, subacute excitement, and, in practically all cases,

¹ Major Hotchkis notes that of the 45 cases of cut throat admitted to Dykebar Military Hospital during a year, 18 were alcoholics. *Journ. of Ment. Science*, April 1917, p. 245.

hallucinations. The history of many of these cases suggested that though alcoholism was a prominent feature in predisposing to a mental breakdown, of still greater importance was the strain and stress of the campaign, and had it not been for this the breakdown would either never have occurred, or would have been postponed."¹

Some of the cases have exhibited comparatively slight maniacal symptoms but without hallucinations. At times they have been violent; but the condition has not been that of the furious violence and the transient clouding of consciousness of *mania a potu*, although it is difficult to say how much of the trouble is due to alcohol and how much to instability aggravated by the excesses. In some of them, however, there appears to be no doubt that there is defectiveness and that the taking of alcohol in large quantities is only a symptom of the underlying disorder.²

In addition to the more acute cases where alcohol is the chief factor in upsetting the balance there have come under observation others exhibiting the symptoms of chronic alcoholic insanity. These have been for the most part among the older men—frequently members of the Labour Battalions—many of whom doubtless had the condition fairly well developed before enlistment. Many of these, too, have been employed at the base where alcohol could be obtained with comparative ease. Stress may have had something to do with the accentuation of the trouble; and it is certain that some of them could not have failed to attract attention to their morbid condition had this been as obvious when they were sent out as it was when they returned. In the majority of these cases the symptoms were delusions of persecution and auditory hallucinations. Others were influenced by the ideas of conjugal infidelity so habitual in chronic alcoholic insanity. Amnesia and paramnesia were also common symptoms.³

It is obviously impossible to speak with any degree of certitude

¹ "A War Hospital for Mental Invalids," p. 245.

² "Chez d'autres sujets, prédisposés vésaniques, cela va de soi, c'est un *délire vésanique* qui se greffe plus ou moins précocement sur la psychose alcoolique aiguë et qui, celle-ci disparue ou améliorée, évolue ultérieurement pour son propre compte." Of the chronic persecuted cases the same writer remarks that they are "plus persécutés, en réalité, qu'alcooliques."—Régis, "Précis de Psychiatrie," p. 583.

³ "Fabulation," according to some observers, is the rule and not the exception among others than the alcoholic patients!

as to the general effects of alcohol in bringing about these mental breakdowns. The matter is so complicated, and accurate information as to the amount of alcohol taken is so difficult to obtain, that one can only form a general impression of its influence. Statistics in regard to the effects of alcohol are only satisfactory to those who wish to be convinced in some particular direction. It does appear to be the case, however, that it is a potent factor in bringing about mental disorder: and when one considers how greatly periods of stress increase the demand of the nervous system for some exogenous stimulation, it is easy to conceive that alcohol has exercised no slight effect in this campaign.

MENTAL DEFICIENCY.

Mental defect is notoriously a term difficult to define. Its connotation for the most part varies with the idiosyncrasy of the definer. It is true that from moral defect with—quite frequently—more than average mental ability down to imbecility, there are many gradations of intelligence: yet it is not, on the other hand, justifiable to judge all men too hypercritically. Even granting this, it is difficult to understand "how some of them passed the recruiting officers." It is probably true, as the same writer remarks, that "the worst types got in during the first rush of recruits under the voluntary system";¹ but this does not by any means suffice to explain many other cases. To some extent it has been due to inability to appreciate what mental defectiveness really is; but a still more likely explanation is that, even when the recruit was seen to be a mental defective, it was considered that military training would "teach the young idea how to shoot"—literally and metaphorically! It is obviously not realised that, to quote the old Scots saying, "one can't make a silk purse out of a sow's ear"; in other words, that the trouble is one of the quality of the material structure with which we have to deal. The brain-cells are defective and no amount of training can bring their function—or "intelligence"—to a high level of efficiency. It is an error which still pervades only too greatly our whole educational system.

When one has seen a large number of the cases which have been sent home it is easy to understand that many of them might

¹ "A War Hospital for Mental Invalids," *loc. cit.*, p. 245.

well become "not only useless, but often positively dangerous to their comrades," and to appreciate the full force of the instance given by the same writer of the man on sentry-duty who, on being asked by his officer what he would do if the enemy appeared, replied that he would present arms and say, "Pass, friend, all is well"!¹

On the other hand, it is remarkable how much of the stress and strain of active service some of those with quite a high degree of mental defectiveness have withstood before breaking down. Even, however, if these had been more numerous than is actually the case it would be dangerous to establish a precedent thereupon. In times of peace these defectives might be utilised without any great danger to the machinery: and even in war-time they could with careful and deliberate selection be fitted into their proper places in the military organisation. But when expedition is necessary, only the most plastic material can be made use of; and this cannot be found in the mental defective on the one hand, or, on the other, among those whom advancing years have rendered incapable of adaptation.

What has been said does not, of course, apply to those cases where the defect is only slight, and, therefore, not to be gauged by the ordinary methods of examination, nor again to those where it is of the nature implied in the term "moral." Quite a fair proportion of these have come satisfactorily through the ordeal. On the other hand, it is precisely from their ranks that have come those individuals who have been almost a constant source of trouble during the time they have been in the army. Their conduct-sheets are well filled with the record of their offences: and from first to last they have wasted the time and energy of their official superiors. It is among these cases, too, that it is, at times, difficult to discriminate between madness and badness; many are criminals, potential if not actual.

When, however, these defective cases have been tried and found wanting, the best method of procedure appears to be to discharge them as unfit for further service. As has been said, it is hardly feasible, considering the immense numbers of men who are being dealt with by the military organisation at the present time, that the comparatively few who are ineffective for general purposes should have special arrangements made for them. It

¹ "A War Hospital for Mental Invalids," *loc. cit.*, p. 245.

is probably better, therefore, to allow them once more to gravitate to their ordinary level in the social system, and that as quickly as possible.

Among the mentally defective there occurred, as was to be expected, attacks of insanity of varying degrees of acuteness—mania, melancholia, and confusion. These conditions obscured for a time and in certain cases the underlying defect which only became apparent when the acute symptoms passed off. It may be that the patients were left more mentally deteriorated than they would have been had no such acute attacks occurred: but that is a question as difficult to decide as it is in every case of acute insanity where apparently entire recovery takes place, or as it is in other bodily disorders where function is re-established after, for example, cloudy swelling in the cells of a particular organ. Where dubiety exists in this way it is only fair—in cases where there is evidence of exposure to stress—to presume that the defect has been aggravated.

IV.

Prolonged rest is necessary in order to allow recuperation to take place. If the cells have deteriorated it is obvious that a considerable period must elapse before they become rehabilitated sufficiently to enable them to carry on their functions. An absence of stimulation permits this, and, at the same time, an undue strain is not thrown upon the portions of the brain which are still functioning. It does not seem wise, therefore, simply to attack certain symptoms such as amnesia and mutism where these are merely subsidiary to a more generalised defect. Speech is regained and memory improves as the general state ameliorates. Rest and quiet are, therefore, of prime importance, at all events in the early stages. Noxious stimuli—and among these may be reckoned worrying relatives—should as far as possible be eliminated. Even the importunate doctor should not unduly harass the patient's tired brain! This is, of course, more necessary in confusional conditions: the stuporose patient is impervious—or apparently so—to ordinary stimuli. It goes without saying that everything must be done to improve the general health: and diet tonics, &c., are important accessories. Very much the same may be said in regard to neurasthenics. Rest, possibly for a time even confine-

ment to bed in a separate room, is likely to be of benefit, but this may be, and often is, overdone. Such patients do not need to be fussed over: and, if they are, undue concentration of attention upon their condition tends to perpetuate or even to aggravate the morbid state. It appears also that the constant association of the neurasthenic individual with others who suffer in a similar way is generally prejudicial to his—and to their—recovery; but this is less noticeable when, the general health having improved sufficiently, participation in amusements or occupation of some other kind prevents the accustomed pastime of the valetudinarian—a causerie on symptoms.

When the general health has improved there can be no question that work in the open air is very beneficial. Indeed for very many cases the regimen of the Pythagorean school cannot be improved upon: and is more likely to bring about a cure than certain pseudo-scientific methods which, under a cloak of novelty, are really recrudescences of mediævalism!

Some observers appear to attach importance to the psycho-analytic method. I have no experience of its utility. It may be useful in certain types of case, but not in such as I have had under my care. The neurasthenics and psychasthenics—I am not quite sure of the exact connotation of the term neurasthenia, still less of psychasthenia—may respond satisfactorily to the process of mental excavation; but practically all the cases which I have seen have recovered under ordinary methods of treatment. It is not apparently beneficial in actual mental disorder—and the great majority of cases admitted to this hospital¹ come into this category. It is difficult to see how the theories of this school differ—at least to any appreciable extent—from those of their many predecessors who have based their systems on mental, as opposed to physiological, findings; or, as it might be expressed without injustice, verbal instead of actual.

In regard to cases of acute insanity, they must be treated as such; that is to say by the ordinary methods. Temporising may lead to disaster, and in any case it is doing less than justice to the patients. It must be recognised—however desirous people may be of glozing over the fact—that certain of these patients are insane, just as other individuals have been insane before them. The therapeutic methods at present in vogue have been found

¹ The County of Middlesex War Hospital.

successful in very many cases; and the recovery rate will compare favourably with that in many other diseases to which a greater amount of attention has been directed. It is easy for those who have no practical knowledge of mental disorder—and only for them—to maintain the greater efficacy of their particular nostrum; but it is shrewdly to be suspected that even their views would alter if they were brought face to face with the actual problem of how best to deal with the insane. Many therapeutic systems have thrived on those cases which get well if left alone—or in spite of what is done for them.

It is necessary to segregate acute, noisy patients; and this can only be done where adequate accommodation is obtainable. The padded room is of great utility where the patient is inclined to roll restlessly about on the floor. By the use of it it is possible to obviate the necessity of orderlies struggling with the patient or of resorting to mechanical restraint. It is to be remembered, too, that many a patient will continue to struggle so long as he is manually restrained; while he will cease to do so when left quietly by himself in a room. This has been exemplified in several instances. Patients who have been restrained by mechanical means prior to admission have not required the adoption of such methods after they have been admitted.

For the proper care of these patients, it is essential to have specially trained orderlies. It is needless to labour this point. Those upon whom the responsibility rests of arranging for the supervision of the insane will fully appreciate the reasonableness of this statement. The attitude of the untrained towards mental patients is not generally conducive to the amelioration of their condition. Suicides and accidents are not unlikely to be the results where those in attendance—however willing they may be—have not been accustomed to the care of the insane. The necessary knowledge is not gained in a few weeks; still less can it be evolved—even in the medical man—out of the inner consciousness.

By some it is argued that nothing should be done to suggest to the patient that he is mentally deranged; and many ineptitudes have been uttered by those who, blameless of any taint of practical knowledge of the insane, would discard everything which experience has proved to be of utility in these matters. Nothing is, however, more likely to inspire patients with suspicion than the

often ill-concealed mistrust shown towards them by those who regard every one labelled "mental" as a potentially desperate character.

V.

The various symptoms observed in these patients tend still more to controvert the almost dogmatic assertion that cases of nervous disorder can be differentiated into the categories of "functional" or "organic." That is unless by the word "functional" there is implied disorder dependent upon a less degree of interference with structure than is present in "organic" conditions. But this is not the usual implication of the term, which is used—as far as one can gather—in much the same way as is the word "mental," in what may be described as the metaphysical sense. Function is, however, merely the term applied to the functioning of, for example, the lungs, the heart, or the brain. It is not an abstract something which produces a result: it is itself a result. That functioning of a particular kind produces results which are again the causes of still further results, does not invalidate the argument. The change in the particular structure involved may be transient, and possibly, could it be investigated, might elude the most careful search. Unless we are convinced that our methods of investigation are infallible—which we certainly are not—we must admit the possibility of cell changes. To assume that there are no such alterations in structure and then placidly to ignore even the small amount of material available for investigation is a method little likely to result in the advancement of knowledge. Fortunately there are investigators who have not been satisfied with such obscurantism, and it seems certain that we shall receive still more enlightenment from their researches.

Crile has demonstrated by laboratory experiments that "in an animal driven strongly by emotion" there is a "mobilisation of the energy-giving compound in the brain-cells, evidenced by a primary increase of the Nissl substance and a later disappearance of this substance and the deterioration of the cells."¹ The cell changes due to the emotions are "so similar, and in extreme conditions approach so closely to the changes produced by disease, that it is impossible to say where the normal ceases and the

¹ Crile, "Origin of Emotions," p. 138.

abnormal begins."¹ Such evidence as this should make clear how difficult—indeed, how impossible—it is to divide cases clinically into functional and organic with any degree of certitude. At what stage of change in the Nissl granules does the disorder become organic? If it is argued that in functional nervous conditions no cellular changes have taken place, nothing further can be said except that it is an assertion of a mere hypothesis which lacks proof, and that the evidence so far collected does not support it.² These cases give additional proof—if it be needed—that neurology and psychiatry are not two distinct subjects, though there is too often a quite unnecessary alienation of the one from the other. The two terms really connote different aspects of the same subject—however much the "psychic" school may dissent from this attitude. In no class of case is this more obvious than in those designated hysterical. In these it is more and more apparent there is, among other changes, increased resistance to the passage of stimuli in certain nerve areas. Along with this there may be changes in other parts of the nervous system and, consequently, psychical symptoms. Because certain stimuli—incantations and various methods of "spiritual healing"—have been sufficient to overcome resistance in these cases they have been looked upon as the way in which to bring about a satisfactory result. But the stress and strain of war having increased the number of such cases of disorder and having rendered the problem of treatment more urgent, there has been a corresponding increase in the amount of attention devoted to etiology and to therapeutics. If, however, certain prominent symptoms are only indications of a more or less generalised exhaustion of the nervous system it is obvious that attention should not be unduly concentrated upon them, but that by means of rest and other appropriate measures the condition of the whole organisation should if possible be ameliorated. It may be that rehabilitation will be accomplished by these measures

¹ Crile, "Origin of Emotions," p. 111.

² "For practical purposes it is convenient and serviceable to divide cases into organic and 'functional,' but more than one case has come under my notice that has clearly been a sort of 'missing link' between the two groups. . . . 'Functional' symptoms may be the expression of minimal organic changes."—"On Concussion Injuries of the Visual Apparatus in Warfare, of Central Origin," by S. A. Kinnier Wilson, M.D., &c., *Lancet*, 7th July 1917, p. 1.

alone. If, however, there still remains some obstinate symptoms it appears to be evident that this can be dealt with satisfactorily by means directed particularly towards eliminating it.¹

The war has apparently produced no new nervous or mental disorders. It has increased the number of such cases, and has thus caused some of them to be brought to the attention of many observers who would not in the ordinary course of events have taken cognisance of them. The difference is thus more in the perspective from which they are regarded than in the conditions themselves. Neurasthenia and "shell-shock" have provided ample scope for study—and for the formulation of more or less plausible theories as to their causation. Both terms have been used loosely and in a vague way. It yet remains to be proved what the basis of these conditions is. Further investigation of the function of the ductless glands may throw light upon the part which they play in the matter. In the meantime it is interesting to note—as Crile has pointed out—the extremely suggestive nervous condition of patients suffering from Graves' disease, and the general influence of disordered function in the thyroid gland. Much ground has to be covered, however, before satisfactory conclusions can be reached. It is not yet settled—if we may judge by the conflict of opinion—what is the best method of proceeding to investigate certain of these problems. That being so we may look for still more darkening of counsel; and one of the most certain results to be anticipated is an accession to our terminology if not a corresponding increase of our knowledge!

Note.—Some other matters, such as the possible production of epilepsy by injury and shock, the effects of head injuries in giving rise to mental disorder in certain cases, &c., it was intended to include in this paper. It has, however, already grown to such inordinate length that their consideration must be deferred in the meantime.

¹ See in particular an extremely illuminating paper by Captain Adrian and Dr Yealland on the work done by them at the National Hospital—"The Treatment of Some Common War Neuroses," *Lancet*, 9th June 1917, pp. 867-872; and "Les Psychonévroses de Guerre," by Roussy and Lhermitte (Paris, 1917), p. 158 *et seq.*

Abstracts

BRAIN, SKULL, &c.

ON THE NUCLEUS OF THE OCULO-MOTOR NERVE. (Con-
(236) *tributo allo studio del nucleo dell' oculo-motore comune.*) E.
CAVAZZARI, *Riv. di patol. nerv. e ment.*, 1917, xxii., p. 239.

A MAN, aged 30, claimed exemption from military service on the ground of nervous and visual disturbances. Examination showed paralysis of the sphincter iridis, ciliary muscle, superior rectus, and internal rectus of the left eye. The inferior rectus and inferior oblique were intact. The fundus was normal. On inquiry it was found that this condition had developed suddenly at the age of 14, with headache, bilateral nystagmus, and ptosis of the upper lid, which disappeared, leaving the eyes described above.

The Wassermann reaction was negative, and there was no evidence of meningitis or compression.

The condition is attributed to a central lesion, which was most probably a hæmorrhage. The involvement of some muscles and the escape of others is explained by the fact that the oculo-motor centre is divided into several nuclei, among which those of the intrinsic muscles of the eyes and those of the superior and internal rectus are in close juxtaposition, while the nuclei of the levator palpebræ and inferior rectus, and still more so that of the inferior oblique, occupy an excentric position. J. D. ROLLESTON.

THE SYNDROME OF THE POSTERIOR LACERATE FORAMEN.
(237) (*Le syndrome condylo-déchiré postérieur.*) G. CHAZALET, *Thèses de Paris*, 1916-17, No. 132.

A RECORD of nine cases collected from the literature, occurring in wounded soldiers, of the syndrome first described by Vernet (*v. Review*, 1917, xv., p. 194), consisting in paralysis of the last four cranial nerves. The etiology may be exo- or endocranial, the symptoms in the former case being due to traumatism by a projectile or other wound in war, and in the latter to a new growth, tuberculosis, and especially syphilis. J. D. ROLLESTON.

THE SYNDROME OF THE POSTERIOR LACERATE FORAMEN
(238) **OF ENDOCRANIAL ORIGIN.** (*Syndrome condylo-déchiré postérieur d'origine endocranienne.*) J. A. SICARD and L. RIMBAUD, *Paris méd.*, 1917, vii., p. 209.

A RECORD of a case in a soldier, aged 24, who presented paralysis of IX., X., XI., and XII. on the left side (*v. Review*, 1917, xv., p. 194). The condition was probably due to a meningo-radicular process in

the left half of the cervico-bulbar region, and was probably syphilitic in origin, owing to the excess of albumen and cells in the cerebro-spinal fluid and the success of specific treatment.

J. D. ROLLESTON.

IMMEDIATE ONSET OF REMOTE NERVE LESIONS IN
(239) **WOUNDS OF THE HEAD.** (*Lésions nerveuses à distance et immédiates dans les blessures de la tête.*) H. GOUGEROT, *Paris Méd.*, 1917, vii., p. 301.

SOME war wounds, especially in the head, give rise to nerve lesions which do not have any obvious connection with the course of the projectile, *e.g.*, a man wounded in the face may develop right hemiplegia with aphasia. Such cases may be wrongly diagnosed as hysterical. They must be distinguished from the remote nerve lesions of late onset due to infective foci and nerve degeneration following contusion or compression, as well as from the reflex nervous disorders described by Babinski and Froment. Gougerot records two illustrative cases.

1. A soldier was wounded by a bullet, the entry of which was in the left cervical region and the exit in the left malar region. Right hemiplegia and aphasia developed immediately after he had killed the German who had shot him. Lumbar puncture showed the presence of meningeal hæmorrhage. Improvement took place, spastic brachial monoplegia, right lower facial paresis, and aphasia only persisting.

2. A soldier was hit by a shell fragment which penetrated the right half of the upper lip and lodged in the left submaxillary region. Bilateral facial paresis and paralysis and anæsthesia of the tongue developed. Gougerot attributes this condition to peripheral lesions of VII., IX., and XII. by contusion. J. D. ROLLESTON.

ATTACKS OF HYPOTHERMIA AS AN EPILEPTIC EQUIVA-
(240) **LENT IN A TREPHINED PATIENT.** (*Crises d'hypothermie comme équivalent épileptique chez un trépané.*) P. CARNOT and A. DE KERDREL, *Paris méd.*, 1917, vii., p. 395.

A SOLDIER, aged 24, who had been trephined in the frontal bone as the result of compression of the cerebral convolutions by retraction of the scar, developed (1) attacks of Jacksonian epilepsy characterised by violent pain and spasmodic contractions of the right arm. (2) Periods of hypothermia of several days' duration preceding or taking the place of the sensori-motor attacks.

The rectal temperature in both periods ranged from 93.5°-95.7° F.

Both the hypothermia and the Jacksonian epilepsy disappeared simultaneously as soon as the compression caused by the scar had been removed by operation.

J. D. ROLLESTON.

- ON A CASE OF SPASTIC PARAPARESIS FOLLOWING A GUN-**
 (241) **SHOT WOUND OF THE PARIETALS IN THE NEIGHBOUR-**
HOOD OF THE BREGMA. (Sopra un caso di paraparesi
 spastica consecutiva a ferita da proiettile dei parietali in
 vicinanza del bregma.) C. FRANK, *Riv. di patol. nerv. e ment.*, 1917,
 xxii., p. 441.

A SOLDIER, aged 30, as the result of a tangential gunshot wound of the two parietal bones on August 1915 in the mid-line of the cranium, in the immediate neighbourhood of the bregma, developed tetraplegia and motor aphasia. After removal of some bony splinters in the area of the fracture, the paresis of the upper limbs and the aphasia rapidly improved, so that by November all disturbance in the limbs and speech had disappeared. On the other hand, the paraplegia in the lower limbs persisted until the end of November, when it became transformed into a spastic paraparesis most marked in the distal segments (feet and legs). The paresis was most marked on the right, *i.e.*, on the opposite side to the greater lesions of the cranium.
 J. D. ROLLESTON.

- CONTRIBUTION TOWARDS THE STUDY OF CEREBELLAR**
 (242) **LOCALISATIONS IN MAN: A COMPLETE CEREBELLAR**
PARASYNDROME DUE TO A WOUND OF THE INFERIOR
VERMIS (THE SYNDROMES OF DUCHENNE AND OF
BABINSKI, ASSOCIATED AND LIMITED TO THE LOWER
LIMBS). (Contribution à l'étude des localisations cérébelleuses chez l'homme: parasyndrome cérébelleux complet par blessure du vermis inférieur (syndromes de Duchenne et de Babinski, associés et limités aux membres inférieurs).) ANDRÉ LÉRI, *Bull. de l'Acad. de Méd.*, 1917, 3 Sér., lxxvii., 8 Mai, p. 596.

A MAN of 31 was wounded in the neck by two bullets: one was superficial in the left occipital region, the other deeper just to the left of the middle line. There was opisthotonus which soon passed off, and gradual improvement in gait. Six weeks later examination revealed Duchenne's cerebellar syndrome, titubation, antero-posterior tremor of lower limbs, and vertigo, with a tendency to inclination to the left in antero-posterior displacements of the head; also Babinski's syndrome, viz., typical asynergia and adiadicocinesia of the lower limbs, with some degree of cerebellar catalepsy, but no hypermetria: no atonia, ataxia, or objective sensory changes: lower limb reflexes brisk, especially on right side. In a few months all these symptoms almost entirely cleared up. The writer concludes that a superficial lesion of the inferior vermis can give most of the symptoms of the two

cerebellar syndromes of Duchenne and Babinski respectively ; and that in the upper or middle part of the inferior vermis there is a centre for co-ordination of movements of the lower limbs. He also claims that an analysis of his patient's symptoms justifies the following conclusions :—

1. Titubation is totally independent of atonia and asthenia, and also of vertigo.

2. Asynergia, adiadococinesia, and tremor can be produced without any excessive movement ; hypermetria, which is one of the important elements of Babinski's syndrome, is therefore not the determining and necessary cause of asynergia, adiadococinesia, and tremor ; and it is wrong to attribute these symptoms, as some writers have done, to dysmetria or hypermetria.

3. Vertigo, of cerebellar origin, can be the consequence of displacement of the head in a single direction, *e.g.*, the sagittal ; it seems therefore probable that the cerebellar cortex has distinct relations with each of the semicircular canals.

Opisthotonus can follow a lesion of the inferior vermis of man, as of an animal.

LEONARD J. KIDD.

THE TEMPERATURE AS A VALUABLE GUIDE TO DIAGNOSIS, (243) PROGNOSIS, AND SURGICAL TREATMENT IN CRANIO-CEREBRAL TRAUMATISMS. J. W. COURTNEY, *Boston Med. and Surg. Journ.*, 1917, clxxvii., Oct. 11, p. 511.

COURTNEY claims that of all the diagnostic and prognostic manifestations of intracranial traumatism, the temperature throughout stands supreme in the scale of importance. A depression of cardiac force is no more significant here than in other forms of injury or disease. A practically normal or a full and relatively slow pulse is encountered as often in recovering cases as in those that do badly. Respiration is usually a most unreliable measure of the degree of danger that menaces, except in initial shock or in the closing phase of a fatal case. In intracranial hæmorrhage, shock and subnormal temperature are the immediate effect. But unless the bleeding is very extensive and complicated by co-existing alcoholic intoxication, the temperature soon regains the normal, and is seldom elevated more than a degree throughout the whole course, provided no great degree of contusion and none whatever of laceration be present. In most cases of uncomplicated contusion, temperature rises quickly from the subnormal level of shock, and may go as high as 102°. Almost from the beginning its recessions are striking: it shows a daily nocturnal rise. In fatal cases it may go very high. In brain laceration the initial fall of temperature from shock is of extremely brief duration. It

makes little practical difference whether the laceration occurs in alleged heat-producing centres or elsewhere, or whether it is accompanied by extensive bleeding. In all cases temperature rises: in bad cases it may reach 104° or 105° in a few hours. While recessions may occur, the fact remains that early and persistently high temperature in cranio-cerebral traumatism invariably denotes brain laceration. In the later complications of intracranial injuries—notably in arachnitis and abscess—the behaviour of the temperature is equally constant. Chills during the course of a case of head injury do not indicate pus formation: they are rarely seen during the course of a practically uncomplicated contusion: and in arachnitis they are mainly conspicuous by their absence. An arachnitis is generally unrecognisable for several days after a trauma: its onset is marked by a sudden jump in temperature, with such signs of cortical irritation as delirium, somnolence, or great motor restlessness. The initial rise may be as much as 6° in a few hours: it is followed by curious but characteristic fluctuations, not only from day to day but from hour to hour: there may be a difference of 4° between the highest and lowest records of any twenty-four hours. The average temperature may be as high as 103° plus. In cerebral abscess the temperature is commonly normal during a sinister period of quiescence which follows the antecedent activity of the process that favours the growth of pyogenic organisms, and subnormal after the abscess has caused local manifestations of its presence. But abscess may appear before disappearance of signs of an antecedent process; and in this event it may continue high to the end. As to prognosis—when the temperature chart indicates that a given compression of brain is due to a hæmorrhage which is practically uncomplicated, prompt ligation of a bleeding vessel and turning out of a clot will save life. When it indicates contusion, we are dealing with the most insidious and treacherous of all cerebral traumatisms: the prognosis must here always be guarded even when neurological symptoms appear slight: only in the mildest cases of contusion can danger to life be held to be over before the end of the second week. In cases of laceration, trephining is not even to be thought of. It is not invariably fatal, and even a temperature of 105° does not preclude recovery: in favourable cases convalescence may be painfully slow, and temperature stand at 99° for a long time. In every case of arachnitis the outlook is dark. In abscess it hinges largely on the matter of accessibility. Directly an abscess is evacuated temperature is apt to rise a point or two, and remain somewhat elevated for a day or two. Courtney, towards the end of his valuable paper, remarks that “while close observation of tem-

perature behaviour in cranio-cerebral injuries markedly broadens our understanding of the intimate nature of their underlying pathology, it at the same time automatically betrays the extreme narrowness of the field of indications for legitimate surgical intervention in these injuries."

LEONARD J. KIDD.

TREATMENT OF CRANIAL INJURIES IN WAR. J. ANDERSON, (244) *Brit. Med. Journ.*, 1917, ii., July 14, p. 42.

THIS paper is a plea for early and complete operative interference in gunshot wounds of the skull. All scalp wounds, except minute abrasions, ought to be excised. A skiagram should be taken in all cases. A number of points in connection with the operative technique are discussed.

A. NINIAN BRUCE.

SPINAL CORD.

A PLEA FOR THE NON-CATHETERISATION OF THE URINARY (245) BLADDER IN CASES OF GUNSHOT WOUNDS OF THE SPINAL COLUMN. FREDERIC A. BESLEY, *Journ. Amer. Med. Assoc.*, 1917, lxi., Aug. 25, p. 638.

IN a certain general hospital containing 1,500 beds not a single case of spontaneous rupture of an over-distended bladder occurred. Logic and experience teaches the writer that in cases of injury of the spinal cord the bladder should not be emptied by catheterisation, unless there be a pathological stricture of the urethra, which would prevent overflow. He has never seen any deleterious effects from over-distension of the bladder in spinal cord cases that have not been catheterised. In one case of benign spinal cord tumour, which was removed, the bladder was for two years distended up to the umbilicus, and yet the patient regained complete control of her bladder, and never showed any evidence of kidney disturbance or injury. Besley concludes that it is almost impossible to catheterise the bladder in a case of gunshot wound of the spinal cord and column, accompanied by paralysis, without producing an infection and subsequent inflammation of the genito-urinary tract. Infection rarely, if ever, occurs without catheterisation. Distension of the bladder, allowing it to overflow, is not harmful to bladder or kidneys. Many patients suffering from an injury to the spinal cord and column may ultimately be saved by surgical operation, if they do not succumb to an early cystitis and purulent pyelitis.

LEONARD J. KIDD.

SOME OBSERVATIONS IN SPINAL CORD SURGERY. HAROLD (246) NEUHOF, *Annals of Surgery*, 1917, lxx., April, pp. 410-437.

THE writer has had unusual opportunities for the prolonged observation of the effects of spinal cord lesions, principally traumatic ones, and the effects of various operative procedures on such lesions. In this valuable paper his observations are grouped under various headings; some of these are related, but are separately presented to bring out certain points.

1. *Fascia Transplantation into Defects of the Spinal Dura.*

The problem of dealing with large defects of the dura, the necessary result of its sacrifice at operation, has not been adequately solved. With reference to the procedures adopted by two sets of surgeons, viz., (1) avoidance of suture of the dura, and reliance on an accurate closure of musculature and aponeurosis in order to prevent leakage of cerebro-spinal fluid, and (2) the covering of the defect by Cargile membrane, which is expected to remain securely in place, and about which adhesions are supposed to develop, Neuhof believes that whatever clinically satisfactory results follow such treatment of spinal dura defects depend on the careful approximation of the layers of the wound, and not on the implanted animal membrane. Experimentally and clinically, defects of the cerebral dura have been adequately replaced by free autoplasmic transplantation of fascia lata. In a series of experiments Neuhof was able to demonstrate that defects of the spinal dura can be treated in the same way. In fact, fascia transplantation in the latter region was found to have distinct advantage over its implantation into the cerebral dura, for, in contrast, adhesions between the transplant and the underlying cord were not observed even after prolonged periods (more than one year). In these experiments the inner surface of the transplant sutured into a defect of the spinal dura remains smooth, and becomes lined with a layer of flat, mesothelium-like cells continuous with the dural lining, and the transplant becomes converted into an equally strong resistant layer of altered connective tissue. The correct clinical method of treating large spinal dura defects appears, then, to be by transplantation of fascia. In spinal operations the fascia is close at hand in the shape of the aponeurosis over the erector spinæ; the sacrifice of the necessary part for transplantation is harmless. Neuhof finds that the chances for a successful fascial transplant into a visceral defect are far greater and more uniform if the transplant is sutured into and not over the defect. The technique and the result in a case are described; no leakage of cerebro-spinal fluid occurred, and no bad effects have followed the transplantation after a considerable lapse of time.

2. *The Operative Treatment of recent Spinal Cord Injuries:
Indications and Contra-Indications.*

Whilst almost all surgeons recognise (1) that the present-day surgery for complete crushes of the cord is quite hopeless, and that operation is therefore to be avoided, and (2) that it is very difficult often to differentiate acute œdema, accompanying various lesions, from total destruction of the cord, there is far less uniformity of opinion as to the operative indications for the varying degrees of injuries to the cord short of complete destruction. While some say we should wait for evidences of increasing damage to the cord, others hold one should not wait to see if the patient will recover more or less completely without operation. As a result of much study of these cases Neuhof gives the following reasons for assuming an extremely conservative stand for surgical interference in spinal cord injury, and offers afterwards two indications for operation: (1) In the great majority of cases, whatever damage has been done to the cord cannot be undone by operation. Fragments of bone that have bruised or lacerated the cord have in many instances sprung back so that it is rare to find, at operation, a fragment of bone actually pressing on the cord. Cord injuries may exist in the absence of demonstrable fracture, or with fissure fractures of vertebral bodies and laminae; (2) on operation, partially or completely detached bony fragments are often found in the musculature; in the cervical region particularly these may support the injured column, and their necessary removal for exposure of the cord tends to make collapse of the column more likely; (3) the difficulties in immobilisation after operation are enhanced because of the necessary removal of spines and laminae; an illustrative case is given; (4) operation greatly diminishes the important muscular support of the erector spinae, because of the necessary division and retraction of the muscles, and the subsequent paresis or paralysis; (5) the preparations for operation and the immediate post-operative immobilisation are all fraught with danger of additional damage to the cord out of proportion to what can actually be done for relief in most cases. Distortions of spinal column may result from transference from bed to stretcher and operating table; injury may follow struggling during anaesthetisation—in rolling to the prone posture on the table loose fragments may press on the cord, especially in cervical laminectomy; (6) operation itself is an added danger, for patients are often in poor physical condition; (7) often landmarks are greatly distorted; in not a few cases the position of the cord can only be guessed at, and it suffers further damage by various necessary surgical procedures; (8) the primary manifestations of severe cord injury may clear up in great part

without operation, and there may be only very slight residual evidences of a mild injury to the cord; (9) the immediate after-effects of laminectomy must also be considered, viz., more prolonged stay in bed owing to diminished support to the column after fractures of the vertebral bodies, and resulting complications such as pressure ulcers, anaemia, &c. Neuhof believes that the following two definite indications are the only ones that call for operation for fresh spinal cord injuries in civil practice at the present time: (1) Progressive intraspinal hæmorrhage as indicated by repeated lumbar puncture; (2) unquestionable X-ray demonstration of a fragment of bone encroaching on the spinal cord at the level to which the neurological manifestations point. Neither of these is an absolute indication for operation if the manifestations of cord compression are not severe.

3. *The Limitations of Röntgenography in the Diagnosis of Spinal Injuries.*

It is not universally recognised that injuries of the contents of the spinal canal not infrequently exist in the absence of any lesion demonstrable by Röntgenography; Neuhof refers here specially to cases in which there is undoubted evidence of injury within the dural sac (as shown by presence of blood in the lumbar puncture fluid and neurological manifestations referable to a definite cord level), particularly of the dorsal and lumbar regions; he gives an illustrative case.

4. *The Diagnostic and Therapeutic Value of Lumbar Puncture in Spinal Injuries.*

The great value of lumbar puncture in the diagnosis of spinal injuries lies in the demonstration of the presence or absence of blood in the cerebro-spinal fluid. Cases are detailed in which the discovery of blood in the spinal canal was the only evidence that vaguely suspected spinal injuries were in fact vertebral traumata with cord compression. It may be of very great value also in cases (1) of suspected spinal injury in patients suffering from alcoholism; (2) in comatose or mentally confused cases; or (3) in injuries suspected in cases of hysteria. Other points are brought out in this section of Neuhof's paper.

5. *The Surgical Treatment of Post-Traumatic Deformity of the Spine (Kuemmel's Disease) with Spinal Cord Symptoms.*

More than 100 cases have been reported since Kuemmel, in 1891, first made the affection generally known. It may develop after injuries ranging from mild to very severe, and

from direct or transmitted violence. After describing the three stages in its symptomatology which can usually be recognised, and the commonly adopted treatment by plaster jackets, &c., Neuhof remarks that while immobilisation appears to be the correct treatment for most cases, yet it is not at all evident that the method is equally logical if the late symptoms are referable in part or entirely to lesions of the spinal cord or cauda equina. He advocates operation for these cases, and details three personal experiences.

6. *Indications for and results of Operation in some Post-Traumatic Lesions of the Spinal Cord other than Kummell's Disease.*

Elsberg has recently described various lesions found at operation: narrowing of spinal canal by callus; pressure on cord by displaced fragments of bone, or by one or more vertebral bodies; fracture of a transverse process producing root pain; rupture of ligamenta subflava; and fibrous bands constricting the cord. To these Neuhof adds a description of a few additional types of lesions the late results of injuries, the slighter of which may improve much by well-planned operation.

7. *Pneumococcus Epidural Abscess secondary to Chronic Localised Osteomyelitis.*

Tuberculosis, syphilis, and actinomyces are the causes of the great majority of cases of chronic osteomyelitis of the vertebræ; any of them may result in the formation of an epidural abscess. In the few cases of chronic staphylo- or streptococcus abscess of the spine, symptoms referable to the cord have not been described. Neuhof reports a case, not because it is thought to be a unique or bizarre condition, but as a clinical picture of a late effect of a bacterial infection.

8. *The Significance of the Disappearance of Reflexes and of Retention of Urine after Laminectomy.*

Elsberg noted that after spinal operations the knee jerks were often lost for from twelve hours to several days after operation, and that the ankle jerks were often much diminished for several days. This loss or diminution of reflexes was seen even in cases in which greatly exaggerated reflexes existed before operation. Urinary retention for several days to weeks has been commonly observed after laminectomy even in the absence of pre-operative vesical disturbances. Neuhof believes that transient post-operative disappearance of reflexes and retention of urine are the results of

mild injuries to the spinal cord at operation, owing to the necessary operative manipulations; he gives reasons for this belief, and points out its practical bearings.

9. *The return of Reflexes and of Sensation after Cauda Equina Injury.*

Some observers maintain that the loss of reflexes and of sensation, with corresponding loss of muscle tone and power, are permanent after injury to the cauda equina, and that, therefore, the sole indication for operation for such injuries is the relief of pain. Neuhof presents a case as evidence that this view is erroneous (for relatively recent cauda injuries at any rate) because it demonstrates the prompt return of reflexes, sensation, and muscle power, after an injury which did not sever the caudal roots. (In this case operation was performed four months after the injury; and in three weeks after operation there was excellent muscle tone, normal power of both lower extremities, free range of motion at knees, ankles, and toes, normal jerks, and no residual sensory defects.)

10. *The Significance of Bed-Sores.*

Neuhof ends his paper by insisting that decubitus ulcers are not primarily due to the spinal disease, for, unless they be very extensive, they frequently heal with proper nursing and treatment; this has been his experience even in cases of irremediable spinal affections. It is quite refreshing to read, in the last paragraph, of this very experienced surgeon that "although many advances have been made in spinal surgery in recent years, the whole subject is as yet in an early stage of development." LEONARD J. KIDD.

EARLY TREATMENT OF GUNSHOT INJURIES OF THE SPINAL
(247) **CORD.** H. M. W. GRAY, *Brit. Med. Journ.*, 1917, ii., July 14, p. 44.

ROUGHLY speaking, it may be said that operation is indicated or advisable at a casualty clearing station:—

1. In the presence of incomplete paralysis of motion or sensation below the lesion; especially
2. If X-rays show displaced fragments of bone or the presence of a piece of metal in or near the cord.
3. When the symptoms of paralysis have developed after the infliction of the injury, unless due to inflammation in cases which have been "lying out," when operation is practically hopeless.
4. When pain, due to pressure on nerve roots, is excessive and uncontrollable.

5. In very exceptional cases, when the character of the wound is such that sepsis, although not already evident, is likely to develop and cause rapid death.

In cases retained for more than a few hours, urotropine should be given as a "routine" in an attempt to prevent cystitis.

Operation, in suitable cases, is the same as in trephining the skull in gunshot injuries, both as regards technique and indications for dealing with the dura, &c., except that in the one case we are in contact with inert and in the other with vital portions of the nervous mechanism.

A. NINIAN BRUCE.

A NOTE ON CERVICAL LAMINECTOMIES. With an illustrative (248) case. R. B. BLAIR, *Lancet*, 1917, ii., Aug. 11, p. 200 (Illust.).

A SOLDIER received a shell wound in the neck, posteriorly quarter of an inch to the right side of the middle line, on a level with the interval between the third and fourth cervical spines. When seen ten hours later paralysis of both upper limbs and the right leg was present, with absence of reflexes and severe pain in both arms. X-rays showed a small shell fragment lying against the right lamina of the fourth cervical vertebra.

At the operation the right lamina, depressed about a quarter of an inch, was found to be shattered, and was removed by bone forceps. Voluntary movement gradually returned. The bladder was evacuated by a catheter, and an old stricture in front of the triangular ligament was discovered. It was later thought that under such circumstances a suprapubic cystotomy would have been preferable.

The deciding factors for early operation in wounds of the spine are: (1) incomplete paralysis; (2) accessibility of the muscle; and (3) pain.

A. NINIAN BRUCE.

FRACTURE AND DISLOCATION OF THE SACRUM. RUPTURE (249) OF THE THIRD, FOURTH, AND FIFTH SACRAL NERVES; LAMINECTOMY; NERVE ROOT SUTURE. ANTHONY HARRIGAN and JOSEPH BYRNE, *New York Acad. Med.* (Sect. Surgery), Oct. 5, 1917 (*Med. Record*, 1917, xcii., Oct. 27, p. 741).

A CARPENTER, 44, fell 9 feet from a scaffold, and thence 6 feet to the ground. Just after the injury he had power in the limbs, but no control. Ever since the injury he had loss of control of both bladder and rectum, with absence of erections and of sexual desire. Marked kyphotic deformity, with some scoliosis to right at base of sacrum and a depression at the lower end of the spine on left, the size of a hen's egg. To right of this was the dislocated coccyx. Dr Byrne stated that the neurological examination showed no motor defects except the vesical, rectal,

and genital disturbances noted. The sensory manifestations were pain and soreness in right buttock, and at times a shooting sensation from anus to penis. Sensibility for all forms of superficial stimulation was absent over both buttocks, corresponding to the root of distribution of third, fourth, and fifth sacral nerves. Where, however, the stimuli were of such a nature as to reach the deeper structures, that was pressure touch, compass points, simultaneously and consecutively applied with steady pressure, extremes of heat and cold in prolonged massive applications, the sensibility was preserved even on the area of skin distribution of the third, fourth, and fifth sacral roots on left, although these roots were found completely severed on that side. The glans penis was insensitive to prick, as was the perinæum and scrotum, except on either side bordering the thigh. Bulbo-cavernosus reflexes absent on both sides, also anal reflexes to stimuli applied cephalad of the centre of the anus. Diagnosis made was rupture of third, fourth, and fifth sacral roots on right. Since operation, repeated examination had shown a marked improvement on the sensory loss, especially over perinæum, scrotum, and penis. Genital functions and control of bladder and rectum had returned almost to normal. In discussing the symptoms, Dr Byrne said that the motor mechanism of the vesical and rectal sphincters were functioning irregularly before operation: they were in a state of postural tonus (Sherrington), which could be altered at times. There was a loss of sensibility over the skin supplied by the left third, fourth, and fifth sacral roots for all forms of superficial stimulation, and the retained sensibility even for critical elements reached the deeper structures. These facts warranted a reconsideration of Head's division of the peripheral nerves into epicritic, protopathic, and deep sets. A truer and more useful division was that of superficial and deep critical, and superficial and deep affective systems, the aponeuroses underneath the skin being an important carrier of both central and affective receptors and nerves.

Dr Harrigan thus described the operation he performed:— With patient prone, under ether, an incision 7 inches long was made over centre of sacrum: this was extended above over lumbar spine and below to coccyx. There was a definite kyphosis of the upper part of sacrum, and a dislocation at the right sacro-iliac synchondrosis, causing the posterior surface to occupy an inclined plane. With chisel, gouge, and trephine the central canal of sacrum was opened. In order to establish the identity of the cauda equina, which was in doubt, a part of the laminæ and the spine of the last lumbar vertebra was removed. At the point where the cauda entered the sacral canal it had suffered a displacement or pushing backward, so that at this point its direction

was almost at right angles to that which lay within the lumbar vertebræ. This curving and bending backwards of the cauda was caused by the displacement and fracture of the sacral vertebra. The cauda was completely enmeshed in scar tissue. This was carefully dissected away and the cauda freed. A rupture of left third, fourth, and fifth sacral nerves was demonstrated, confirming Dr Byrne's diagnosis. The distance between the ends of these nerves and the cauda, following complete freshening of the ends, was $\frac{1}{2}$ to $\frac{3}{4}$ inch. Each nerve was sutured to the central cauda equina with a mattress suture of plain catgut, "suture à distance." The coccyx, which was completely dislocated from the sacrum, was removed. Further search showed a large plate of bone detached from the anterior surface of the sacrum and pressing against the piriformis muscle. Between this bone and the sacrum an adventitious bursa had developed: this was dissected away, and the piece of bone removed piecemeal. The overlying muscles were sutured with catgut, a small drain was placed in the lower angle of the wound, and the wound closed. Primary union followed. Findings at subsequent X-ray examinations were also presented, which showed extensive damage to the pelvic portion of the sacrum, and the sacro-lumbar angle much increased, so that one would expect considerable interference with the functions of the sacroplexus, cauda equina, and nerves. LEONARD J. KIDD.

NERVES.

A CASE OF PARALYSIS OF THE EXTERNAL POPLITEAL NERVE DUE TO COMPRESSION BY A GAITER. (Un cas de paralysie du nerf sciatique poplité externe due à la compression par la jambière.) A. MOUCHET and B. T. LOGRE, *Paris méd.*, 1917, vii., p. 303.

A CAPTAIN, aged 33, was admitted to a neurological centre for paralysis of the left lower limb. Examination showed considerable atrophy of the calf and typical R.D. in the antero-external muscles of the leg. On inquiry it was found that the patient had been nearly three weeks in the trenches without having been able to take off his gaiters or his boots. The upper extremity of each gaiter ended just at the level of the head of the fibula, where the external popliteal nerve was found to be abnormally superficial and prominent, especially on the left side. The boot, on the other hand, which was very strong and rigid, had acted as a splint and formed a spontaneous support for the paralysis of the external popliteal nerve, so that the paralysis was not noticed until the boot had been taken off in a rest camp. J. D. ROLLESTON.

- PARALYSIS OF THE RECURRENT LARYNGEAL NERVE IN A**
 (251) **WOUND OF THE BASE OF THE NECK.** (*Paralysie ré-*
curentielle dans une plaie de base du cou.) GUYOT (de Bordeaux)
 and A. D'AURIAC, *Paris méd.*, 1917, vii., p. 217.

SIMPLE paralysis of the recurrent laryngeal nerve is rare, having been found in only 0.35 per cent. of wounds of the head and neck according to the statistics of the Desgenettes Military Hospital at Lyons. The writers record a case in a soldier in whom the right recurrent laryngeal nerve was divided close to its origin by a revolver bullet.

Complete aphasia developed. Absolute immobility of the right vocal cord was found on laryngoscopic examination.

J. D. ROLLESTON.

- CONTRIBUTION TO THE STUDY OF CONTRACTURES OF**
 (252) **REFLEX ORIGIN FOLLOWING TRAUMATISM DURING**
CHLOROFORM NARCOSIS. (*Contributo allo studio delle con-*
tracture consecutive a traumatismi durante la cloronarcosi.)
 M. E. FERRARI, *Riv. ital. di Neuropat., Psichiatr. ed Elettrotet.*, 1917,
 x., p. 201.

A RECORD of two cases in soldiers. The first was that of a slight and superficial wound of the thigh, without lesion of any nerve trunk, but with trophic vasomotor and thermic changes and disturbance of electrical conductivity. During chloroform narcosis there was unilateral exaggeration of the knee jerk and ankle clonus on the affected side. In the second case, which was one of hysterical nature unaccompanied by any changes except a slight degree of hypotrophy due to absence of movement, there was no difference in the behaviour of the reflexes on the two sides during chloroform narcosis.

J. D. ROLLESTON.

- ON SO-CALLED REFLEX PARALYSIS. CLINICAL OBSERVA-**
 (253) **TIONS AND STATISTICAL DATA.** (*Sulle cosiddette paralisi*
riflesse. Osservazioni ed esperienze cliniche e appunti statistici.)
 L. GATTI, *Riv. di Patol. nerv. e ment.*, 1917, xxii., p. 409.

THE syndrome of reflex nervous disorders described by Babinski and Froment should be dismembered, inasmuch as the trophic vasomotor and reflex changes which may result from the wound and the constant immobilisation, are not necessarily associated with paralysis and contractures.

Gatti found that trophic vasomotor and reflex changes were present in wounds of the most varied kind, especially when the lesion had required artificial immobilisation, and had not been accompanied by paralysis or contractures. In his experience some, if not all, of these changes could be produced by artificial im-

mobilisation. As regards the reflex paralysis and contractures, Gatti found that they developed chiefly after slight wounds or even mild frostbite, but not after severe wounds. Psychologically, the patients showed no anxiety to get well, though they were in a perpetual state of obsession as regards their infirmity. The cases were only found among soldiers of the national army who were liable to be sent back to the trenches, and were never present among the Austrian prisoners who presented similar wounds.

Gatti regards the paralysis and contractures as a perseveration in attitudes of defence against pain.

The prognosis is not unfavourable, provided that recourse is not made to the ordinary psychotherapy, which can have no effect upon an impotent will.

Treatment should consist in the tonic treatment of the antagonistic muscles.

In view of the increasing frequency of these forms surgeons should be urged to adopt a preventive treatment, and to mobilise the limbs which adopt the characteristic attitude in spite of the pain caused.

J. D. ROLLESTON.

DISTURBANCES OF LOCAL TEMPERATURE IN A CASE OF
(254) **"REFLEX" PARALYSIS OF THE LOWER EXTREMITY.**

(*Troubles de la température locale, à propos d'un cas de paralysie dite réflexe du membre inférieur.*) SOUQUES, MEGEVAND, NAIDITCH, and RATHAUS, *Soc. de Neurol. de Paris*, Oct. 12, 1916.

THE patient was wounded on 3rd October 1914 in the right ankle by a shrapnel bullet. The lesion was, comparatively speaking, trifling, and after slight suppuration it was healed in a month. Soon after, on commencing to walk again, he found that his foot was turning in, and that he could not prevent this. On examination in August 1916 there was typical talipes equinovarus of the right foot, which could be corrected voluntarily when the patient was at rest, but not when walking; the great toe could be flexed and extended, the others were immobile; the leg movements were all weak, and the muscles very hypotonic. A diffuse diminution of sensibility was noted over the right leg, not corresponding to any ordinary distribution. The deep reflexes were exaggerated on the right; the plantar was either nil, or slight flexion. There was a difference of no less than 5° C. between the temperatures of the soles of the feet, the right being the lower. The authors describe in detail a number of experiments on this hypothermy. Without entering on the vexed question of the pathogeny of these cases, they state that treatment has hitherto been ineffective, and that in their opinion they approach the organic and not the functional type of paralysis.

S. A. K. WILSON.

DEFORMATIONS OF THE HAND FROM NERVE INJURIES.

(255) (*Déformations de la main par blessures des nerfs.*) ATHANASSIO-BENISTY, *Nouv. Icon. de la Salpét.*, 1916-17, Nos. 2 and 3, p. 65.

THE author gives a useful description of the varieties of deformed attitude of the hand and fingers that follow nerve injuries. Photographs and drawings illustrate the complete and incomplete injuries of musculospiral, ulnar, and median nerves in their influence on the hand. The concomitant vascular lesions and their effect are given in detail. An interesting section is devoted to the vexed question of "reflex" paralysis and contracture.

S. A. K. WILSON.

SIXTEEN CASES OF PARATONIC DEFORMATION OF THE HAND FOLLOWING ON WAR INJURIES.

(256) (*Seize déformations paratoniques de la main consécutives aux plaies de guerre.*) LAIGNEL-LAVASTINE and COURBON, *Nouv. Icon. de la Salpét.*, 1916-17, Nos. 2 and 3, p. 81.

By the term paratonic paralysis is signified a condition analogous or similar to the "accoucheur's hand" of tetany. The attitude of the hand is not what is commonly found in ordinary organic palsies, nor, for that matter, in hysterical paralysis; it is a state of hypotonus rather than paralysis, of hypertonus rather than contracture. The authors describe at great length some sixteen cases of the condition, which is of clinical and theoretical interest. The paper deserves consultation in the original, but, briefly, it may be said that while the authors recognise the value of the interpretation of such cases given by Babinski and his collaborators—that the condition is one of reflex paralysis—they nevertheless declare that "concomitant motor disturbances of psychical origin occur in these cases with too great frequency for its importance to be ignored from the point of view of therapeutics."

S. A. K. WILSON.

SOME DEFORMATIONS OF THE HANDS AND FEET IN CASES OF INJURY TO THE NERVOUS SYSTEM.

(257) (*Quelques déformations des mains et des pieds chez les "blessés nerveux."*) LÉRI, *Nouv. Icon. de la Salpét.*, 1916-17, Nos. 2 and 3, p. 121.

THE first group described by the writer is that of œdema of a limb following on constriction. In five cases of this kind the presence of a wound had been utilised by the patient for the surreptitious application of a tight bandage. It is possible, no doubt, that in the first place the original bandage applied by the surgeon or dresser was too tight, and this may have started the œdema. In such cases it is a simple matter to cover the limb with wool and

wrap round that a plaster bandage; the oedema soon disappears. The second group is that of vasomotor and trophic changes, the result of combined lesions of nerves and blood vessels. The author concludes that the vascular lesion is not an indispensable pathogenic element in cases of peripheral nerve injuries with vasomotor and causalgic phenomena, and that latter may occur without any concomitant vascular trauma.

An interesting section is devoted to the appearance of the "pied éfilé" ("tapering foot") in cases of lesion of the internal popliteal nerve. The tapering foot is constant in all painful, causalgic lesions of the sciatic. In some instances it is the sole organic sign of a lesion revealing itself otherwise only by pain.

S. A. K. WILSON.

ON A SPECIAL VARIETY OF SPASMODIC SCIATICA. (Di una (258) particolare modalità di sciatica spasmodica.) O. FRAGNITO, *Ric. ital. di Neuropatol., Psichiatria ed Elettrotel.*, 1917, x., p. 115.

A RECORD of two cases in soldiers. Unlike the cases described by Brissaud, the spasm was not of long duration, nor did it occur at the height of the neuralgic paroxysm as in Oppenheim's cases, but it could always be produced by performing Lasègue's test in two stages (flexion of the thigh on the pelvis, and then extension of the leg on the thigh), or by making the patient fully extend his lower limb. The spasm commenced in the great toe, which became extended while the other toes became flexed, and then the spasm spread to the other muscles of the lower limb. Fragnito's first case was an hysterical individual, while the second was free from any hysterical taint or organic disease.

J. D. ROLLESTON.

CAUSALGIA OF THE SCIATIC NERVE, &c. (Causalgie du (259) sciatique, &c.) DE MASSARY, *Soc. de Neurol. de Paris*, Oct. 12, 1916.

THE patient was a soldier of 35, wounded in March 1916 in the back of the right thigh. Typical causalgia supervened. He was operated on on 6th June. The comes nervi ischiadici was sectioned and sutured; the sympathetic filaments surrounding that vessel were at the same time divided. Slow improvement took place, but not a definitive cure.

S. A. K. WILSON.

REGENERATION IN PERIPHERAL NERVES. EDWIN G KIRK (260) and DEAN D. LEWIS, *Bull. Johns Hopkins Hosp.*, 1917, xxviii., Feb., pp. 71-79 (27 figs.).

IN this experimental study the writers have used their tubulization method by means of auto-transplants of fascia lata on the divided

sciatic nerves of adult dogs. The animals were killed at periods varying from one day to thirty-six weeks after operation. In general, four methods of histological preparation were employed:—(1) Fixation in Bensley's fluid and staining in Mallory's phosphotungstic acid hæmatoxylin; (2) Cajal-Ranson silver impregnation—following Held's pyridine technique; (3) hæmatoxylin and eosin—especially in the study of protoplasmic bands; (4) often the silver preparations were counterstained with safranin.

Summary.—1. In the immediate vicinity of nerve trauma associated with break of continuity there occurs an accelerated hyperplasia of the neurilemmal elements which results in the early formation of protoplasmic bands. These develop in both proximal and distal stump, and tend to bridge the defect. Along these protoplasmic pathways the regenerating axis-cylinders from the central stump pass. Whether they reach the distal stump, and neurotise it, depends largely on the extent to which these preformed conduits have successfully prepared the way.

2. All efficient regeneration of nerve fibres (axis-cylinders) is from the central stump. All regenerating nerve fibres, whether the outgrowth of medullated or of non-medullated axons, are in their early stages non-medullated.

3. All medullation begins proximally and proceeds distally, appearing only in those parts of the new axis-cylinder which have acquired an age of five or five and one-half weeks (in the dog).

(In the discussion following the reading of this paper Professor Howell made the following interesting remarks: "The suggestion, quoted from Clark's paper, that the formation of the embryonic fibres is a process of degeneration rather than of regeneration, is, I believe, open to criticism. I do not see how you can avoid the conclusion that the regeneration is intimately connected with, and dependent on, the growth and proliferation of the neurilemmal nuclei. The augmented activity of these nuclei is the key-note to the whole process of regeneration. The great rapidity with which they multiply as the old fibres undergo fragmentation is in fact an extraordinary phenomenon. As van Gehuchten has pointed out, nothing of this kind is observed in a dead nerve. The underlying conditions which lead to this new growth-activity have not been explained. We may assume as a provisional theory that it is due to some chemical stimulus developed in the process of degeneration of the old fibre or to the formation of specific growth-substances, but so far as I am aware we have no good experimental data in regard to this point.")

LEONARD J. KIDD.

- THE NERVE-TRUNK PINCHING TEST IN OPERATIONS ON**
 (261) **PERIPHERAL NERVES.** (*L'épreuve du pincement tronculaire au cours des opérations sur les nerfs périphériques.*) J. A. SICARD and C. DAMBRIN, *Presse Médicale*, 1917, No. 24, 26 Avril, p. 248.

THE writers carry out this test by means of forceps with smooth edges: gentle stimulation of the nerve-trunk by short small shocks imparted by the forceps or by a kind of tickling by the forceps. This is done above, at, and below the lesion. If there be a response of the muscles innervated by the nerve, we can be sure that axons pass through the cicatricial tissue. Sometimes there is a muscular response by this test when there is a total paralysis and complete R.D. Sometimes the test, when applied above the lesion, gives a response in muscles supplied by a neighbouring nerve. And it may happen that while stimulation above the lesion may be definitely positive, it may be less clear at and below the lesion. It seems that in these circumstances the process of neurotisation has taken a lateral course outside the tract of cicatricial tissue.

LEONARD J. KIDD.

- SOME PRACTICAL NOTES ON THE TECHNIQUE OF CONDENSER**
 (262) **TESTING IN NERVE INJURIES.** FRANCIS HERNAMAN-JOHNSON, *Lancet*, 1917, ii., July 28, p. 117.

THE author points out that the ordinary Faradic coil is only suitable for the roughest work, while the Lewis-Jones condenser may be regarded as an instrument of precision, and may with care give results indicative of a degree of nerve blockage which can only be dealt with by surgery as opposed to a partial or a recovering lesion. The method of testing is described, and consists of setting the voltmeter at 100 volts and noting what length of impulse, or size of condenser, is needed to provoke a just noticeable muscular contraction. The "voltage-duration test" and a method of recording results is described.

A. NINIAN BRUCE.

- ON SUTURE OF NERVES. PRELIMINARY NOTE.** (*Sur la suture*
 (263) *des nerfs. Note préliminaire.*) ALBERT FROUIN, *Compt. Rend. Soc. de Biol.*, 1916, lxxix., Dec. 16, p. 1140.

A RECORD of experiments on seventeen dogs, in which immediate suture of the divided sciatic nerve was performed. By means of very fine needles (No. 14 of Kirby) and floss silk, such as are used in suture of vessels, Frouin has been able quite easily to suture the perineurium without touching the axons of the nerve. In all the animals there has been rapid return of motor power, and after fifteen days they have walked very well on the soles of their feet. In only one were there transient trophic disturbances. In all

there was rapid recovery of the traumatic ulcers which are seen during the early days after operation as the result of faulty attitudes, as walking on the dorsum of the foot. The control animals, whose sciatic nerve was divided, showed severe trophic ulcers, loss of toes or foot, and often infection which proved fatal. The writer holds that the fact of the suppression of trophic disturbances, and of rapid spontaneous recovery from traumatic ulcers, of itself justifies the adoption of immediate suture of a nerve after its division.

LEONARD J. KIDD.

**A SIMPLE METHOD FOR MAINTAINING APPPOSITION OF
(264) DIVIDED NERVES AND TENDONS DURING SUTURE.**

G. LENTHAL CHEATLE, *Journ. Roy. Naval Med. Service*, 1917, iii, July, p. 302 (2 figs.)

CHEATLE uses the finest three-quarter curved intestinal needles and long ones; they are all used ready armed with catgut. The method of their use is as follows: The first armed needle is passed a diameter's length from the edge of the cut surface of one end to the corresponding point in the other; instead of completing the passage of the needle, it is left transfixing the ends, and acts as a support and link by fixing the nerve-ends opposite each other. Then at regular intervals are passed into the nerve-ends four threaded needles, which are also left transfixing the nerve-ends as in the case of the first needle. Thus the nerve-ends are now immobilized except in the longitudinal direction. Then, as a rule, it is better to complete the passage of the first needle, and tie the ends of the nerve in apposition while they are still held transfixed by the other four needles. These needles are treated in turn in the same way; when the time for the passage of the last needle has arrived the completed sutures hold the nerve-ends in sufficient apposition to allow the successful completion of the operation. The same method could be applied, and with advantage, for suturing tendons.

LEONARD J. KIDD.

NITRO-PHENOL NEURITIS. (Névrites nitro-phénolées.) SOLLIER (265) and XAVIER JOUSSËT, *Soc. Méd.-Chirurg. Milit. d. l. 14^e Région*, Dec. 19, 1916; *Lyon Méd.*, 1917, cxxvi., Avril, p. 187.

THE writers have seen a good many cases of chronic retro-bulbar neuritis, with or without other peripheral neuritis, among soldiers working in the manufacture of explosive powders: three cases are detailed. The common history is that after six to twelve months' work in the factory the patient has cramps and tingling in the lower limbs, with simultaneous gradual diminution in visual activity and mistiness of vision; then follows inability to read.

Only after several months longer does he consult the ophthalmologist. There is found green-blindness, paresis or paralysis of accommodation, diminution of central vision, concentric narrowing of visual field, and sometimes a central scotoma. At first there are no ophthalmoscopic changes; then appears an œdematous neuritis, and finally a white atrophy of the optic disc. The pupil reacts normally to light, but not at all, or badly, on attempts to converge. No patient has shown any evidence of tabes, alcoholism, or tobacco poisoning. The noxious agent is one of the nitrophenol series; the writers suspect dinitro-chlorobenzol especially. These workers are constantly exposed to vapours from these powders, which have a vaso-dilator effect; the men themselves describe a cyanotic condition of the skin. The writers advise a trial of vaso-constrictor remedies, such as adrenalin, as preventives. And they insist on the rigorous use of masks, gloves, douches, and disinfection of the hands. LEONARD J. KIDD.

ACUTE FEBRILE POLYNEURITIS. (GORDON HOLMES, *Brit. Med. Journ.*, 1917, ii., p. 37.

THIS condition has been found in all parts of our front as well as from the lines of communication, but no more commonly than in civil life. The onset is usually rapid with fever, followed by pains in the legs and lower part of the back. Paresis then develops, and the arms become similarly but less severely affected. The face muscles, speech, and swallowing may be involved. Muscular atrophy and contractures are rare. The symptoms tend to come to a crisis in about one week, and recovery is slow.

The blood and cerebro-spinal fluid were examined in three cases and proved normal. Treatment is symptomatic. The differential diagnosis from other forms of peripheral neuritis is not difficult. A. NINIAN BRUCE.

"SHELL SHOCK."

THE PSYCHOLOGY OF FEAR AND THE EFFECTS OF PANIC
(267) **FEAR IN WAR TIME.** SIR ROBERT ARMSTRONG-JONES, *Journ. Ment. Sci.*, 1917, lxiii., July, p. 346.

THOUGH very readable, this paper is too lengthy for abstraction.

The author is convinced "that the great and underlying cause in many shell-shock cases is to be found in the instinctive and innate sudden unreasonable fear of the 'unknown' which characterises certain temperaments, often those of superior minds; certainly those whose nervous organisation is highly complex, and it is this type of mind that is soonest subjected to dissociation by fear."

H. DE M. ALEXANDER.

**FUNCTIONAL MUTISM FROM A BOMB EXPLOSION AND
(268) HYSTERICAL MUTISM—THEIR CURE BY ETHERISATION.**

(Il mutismo funzionale da scoppio di granata e quello degli isterici. Loro cura con l'eterizzazione.) D. DE SANDRO, *Riv. di patol. nerv. e ment.*, 1917, xxii., p. 9.

A RECORD of two cases, in a soldier aged 25 and a woman aged 35, in whom complete cure of their mutism took place after three or four minutes' inhalation of ether. The success of the treatment is attributed to the irritation of the respiratory tract in general and of the larynx in particular.

J. D. ROLLESTON.

THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF ABSOLUTE

(269) **HYSTERICAL DEAFNESS IN SOLDIERS.** E. F. HURST and E. A. PETERS, *Lancet*, 1917, Oct. 5.

IN the opinion of the writers, severe deafness following exposure to the noise of a shell explosion is generally hysterical. A lesser degree of organic deafness may be produced at the same time, the result of labyrinthine hemorrhage or of rupture of the drum and consequent otitis, but this organic deafness is never absolute.

The presence of the auditory motor or "jump" reflex—a loud noise causing the patient to jump, or at least to blink—does not imply that hearing occurs. This is a subcortical reflex, and a slight reaction may be noted in most cases of hysterical deafness.

Deafness, unlike all other hysterical phenomena, persists during sleep, and is thus an exception to the law framed by Babinski. A malingerer would certainly be awakened by a loud noise, but not so a case of hysterical or organic deafness.

The diagnostic point upon which the writers lay greatest stress is the presence of a normal vestibular reaction in the hysterical patient, when tested for nystagmus by the caloric or rotation tests. "Organic disease or injury of sufficient intensity to produce severe cochlear deafness is invariably accompanied by loss of vestibular activity."

Hysterical deafness is almost invariably accompanied by mutism. The mutism is readily amenable to treatment, but the deafness is not, as a rule, so easily cured. Cases which fail to respond to simple methods of suggestion may be treated by "operation." Under light anæsthesia, a small incision is made behind the ear, to the accompaniment of a loud noise, such as hammering on an iron plate. Two cases are quoted in which this method of treatment was immediately successful.

DOUGLAS GUTHRIE.

CASE OF TEMPORARY BLINDNESS. F. P. MAITLAND and KENNETH (270) CAMPBELL, *Brit. Med. Journ.*, 1917, ii., Sept. 15, p. 360.

A SERGEANT, aged 49, was admitted to hospital with a temperature of 101·2°, and blind. He complained of some headache. Both fundi were healthy, the media were clear, and no thickening of the retinal arteries could be seen. The temperature slowly fell to normal in eight days, when *pari passu* with the reduction of temperature his vision began to improve, and fourteen days after admission he could see well enough to read a newspaper. He later returned to duty.

The usual causes of blindness unconnected with visible eye changes are hysteria, uræmia, and acute retrobulbar neuritis. The case bore some relation to the last, but the pupils were undilated and normal in action, there was no pain on movement of the eyes, nor pain on pressure applied over the globes.

The case was considered to be due to some toxic condition of the blood, which caused either anæsthesia of the rods and cones of the retina, or of the neurons of the visual cortical centres.

A. NINIAN BRUCE.

ALTERATIONS OF PUPILLARY REACTIONS IN SHOCK, DUE (271) TO BURSTING OF LARGE SHELLS, WITHOUT EXTERNAL WOUND. (Les troubles des réactions pupillaires dans le commotion par éclatement de gros projectiles sans plaie extérieure.) GEORGES GUILLAIN and A. BARRÉ, *Bull. de l'Acad. de Méd.*, 1917, lxxviii., 28 Août, p. 158.

A STUDY of twenty-six cases of recent shell shock: the possibility of even a minimal wound of the eyeball was excluded. The pupillary changes comprised mydriasis; inequality of pupils with or without unilateral or bilateral loss of light-reaction; Argyll-Robertson pupils; or the paradoxical light-reaction. The pupillary changes were commonly transitory, lasting from three to twenty days or longer, and ultimately disappeared. In six of the cases there was xanthochromia of the cerebro-spinal fluid with a slight meningeal reaction. The question is raised whether a xanthochromic or blood-stained cerebro-spinal fluid, rendered toxic by hæmolysis, may not act on the root-fibres of the cranial nerves either on the centripetal or the centrifugal limb of the reflex pupillary arc, or whether small basilar clots may compress the root-fibres. The central origin of these pupillary changes by small intra-peduncular hæmorrhages is held to be unlikely. The writers incline to the belief that most of these cases are of peripheral origin, either a true commotion of the retina or of the nerves of the intrinsic eye-muscles, resulting in a temporary asthenia of one or both of these muscles. LEONARD J. KIDD.

SYNDROMES FROM COMMOTIO SIMULATING ORGANIC
(272) **LESIONS OF THE CENTRAL NERVOUS SYSTEM.** (Quelques observations de syndromes commotionnels simulant des affections organiques du système nerveux central.) PITRES and MARCHAND, *Rev. Neurol.*, 1916, Nov.-Dec., p. 298.

A FEW cases have been published of the development of symptoms of organic type from concussion or commotio without external injury, and the authors bring forward an interesting series in this communication.

In one case a soldier was rendered unconscious by the explosion of a shell at one metre only. On examination three days later his condition was quite typical of acute meningitis. On lumbar puncture the fluid was found to be stained with hæmolysed blood. After a long illness of more than a year, during which mental symptoms supervened, he gradually recovered.

In a second case, subsequently on being blown up and losing consciousness, the patient developed all the usual symptoms of general paralysis. The pupil reactions were normal, but the deep reflexes were absent. The Wassermann reaction was negative in the blood; the spinal fluid was not examined. In other respects, however, mentally and physically, the resemblance was complete. After seventeen months all the symptoms disappeared.

A third patient was blown up and remained semi-conscious for eight days. On examination he showed the ordinary symptoms of cerebellar disease in a typical form. After nine months a cure resulted. The authors also describe cases resembling disseminated sclerosis and tabes dorsalis.

In none of the cases was there any external injury.

S. A. K. WILSON.

CHADWICK LECTURE: MENTAL HYGIENE IN SHELL SHOCK
(273) **DURING AND AFTER THE WAR.** F. W. MOTT, *Journ. Ment. Sci.*, 1917, Oct., p. 467; *Brit. Med. Journ.*, 1917, ii., July 14, p. 39.

THE causes and symptoms of shell shock detailed in this paper were already referred to by the author in the Lettsomian Lectures (*v. Review*, 1916, xiv., p. 216). Additional illustrative cases are given in this paper.

The force generated by 17-in. shells is equal to 10 tons to the square yard, and sudden death without visible injury in these cases is probably due to sudden arrest of the vital centres in the medulla. (A case is referred to in which sudden death occurred after exposure to heavy shell fire with no history of gas or burial. Ruptured vessels were found in the medulla, pons, and corpus callosum, and the condition of the heart and lungs supported the theory of arrest of the cardiac-respiratory centres.)

Shell shock must be diagnosed from malingering and dementia præcox associated with anergic stupor. The spinal, cardiac, cerebral, and gastric types of neurasthenia preponderate, and here suggestion plays a predominant rôle. Sexual neurasthenia is rare.

Increase of pressure of the cerebro-spinal fluid has been found at the clearing stations in true shell shock, and relief was obtained by lumbar puncture.

As to treatment, the continuous warm bath (kept continuously at the temperature of the blood) for a quarter to three-quarters of an hour, or even longer, is very soothing. This bath with hot milk at bed-time may suffice without hypnotics; if such are required, the author recommends mist. paraldehyde 2 oz., with or without trional gr. x.-xv., or 15 gr. each of either pot. brom. or chloral with tinct. opii m. xv., or tinct. cann. ind. m. x. Dial two $1\frac{1}{2}$ gr. tablets. If maniacal, administer hyoscin $\frac{1}{8}$ to $\frac{1}{100}$ gr. hypodermically. Give a nourishing, easily digested diet, and attend to the excretory organs. Severe headache requires the ice-bag, aspirin, or phenacetin.

After the patient has recovered from the more serious condition of shock and the mind is becoming clearer, headaches, dizziness, tremors, feeble circulation, and exhaustion have to be combated with rest in the open air and such a sedative as dilute hydrobromic acid, quinine, and strychnine. Pituitrin is useful in some cases with low blood pressure. Noise should be excluded, and the men should not be left alone, but their minds diverted by knitting, bead-work, net-making, &c. As soon as they are better they can indulge in the usual inside games and attend entertainments. Discipline is essential.

In functional paralysis fixed ideas of disability must be combated, and the assertion that the man will not be sent back again to active service acts as a great tonic. Restore functional paralysis by natural means and not by machines. Encourage outdoor work (agricultural, &c.) in easy graduated stages.

H. DE M. ALEXANDER.

THE PRINCIPLE OF PSYCHICAL ISOLATION IN THE TREATMENT OF FUNCTIONAL NERVOUS DISTURBANCES. (Le principe de l'isolement psychique dans le traitement des troubles nerveux fonctionnels.) LANDAU, *Bull. de l'Acad. de Méd.*, 1917, sér. 3, lxxvii., 29 Mai, p. 701.

LANDAU describes a new mode of treatment which he has found of great value in most cases of functional nervous troubles in warfare: he has thus treated about fifty cases. The patient is brought into the presence of other patients who present similar symptoms, but of an organic nature. A detailed clinical examination of the

patient is then made in the midst of his fellow-patients. If, for example, his case be one of a functional causalgia, the physician reviews briefly the history of the case, its mode of production, and the distinctive signs of these conditions. In the case of a functional palsy of a nerve, the physician discusses openly before all the patients the electrical changes, and then makes an electrical examination of the functional case and of an organic one. As a rule, after a few of these public demonstrations and lectures, and sometimes from the first one, there is benefit to the patient. By means of this kind of procedure Landau claims that a state of "psychical isolation" is set up. He describes it also as a sort of bringing the functional patient face to face with himself. The application of the treatment must to some extent vary with the particular case. Three cases are described to illustrate these points.

LEONARD J. KIDD.

BLOOD PRESSURE AND SURFACE TEMPERATURE IN 110
(275) **CASES OF SHELL SHOCK.** EDITH M. N. GREEN, *Lancet*, 1917, ii., Sept. 22, p. 456 (Illust.).

FIFTY-FIVE men showed on admission a pressure below 120 mm. Hg., and of these 25 were between 88 and 110. These were all severe cases. Of the other 55, 28 were between 130 to 150 mm. Hg., and 27 between 120 and 130. Of those above 130 only 4 were severe cases. With the exception of 8 men, all showed subnormal surface temperature, varying from 18° to 31.5° C. All of the cases with a very low blood pressure were suffering from dreams, which woke them in a state of terror, sweating and trembling. Their hands were dusky and clammy, and most of them had a tremor. They showed a marked fatiguability and irritability; most of them were depressed, and showed a lack of self-confidence and initiative; all suffered from headache. On admission nearly all had dilated pupils.

Of the men with a blood pressure above 120, only 10 complained of nightmares, and in these the signs of fear were not so marked. In all cases, whatever the blood pressure, headache was a common symptom. An improvement in the general condition was coupled with a gradual rise of pressure.

A. NINIAN BRUCE.

ON THE NON-EXISTENCE OF NERVOUS SHELL SHOCK IN
(276) **FISHES AND MARINE INVERTEBRATES.** ALFRED GOLDSBOROUGH MAYER, *Proc. National Acad. Sciences of U.S.A.*, 1917, iii., Oct., p. 597.

MAYER's experiments at Tortugas, Florida, during the summer of 1917, indicate that the nervous system of fishes and invertebrates are remarkably resistant to the injurious effects of sudden

explosive shocks transmitted through the water. Many experiments were made on the *Scyphomedusa Cassiopea Xamachana*. The medusæ were paralysed by removing their marginal sense organs, and then a ring-shaped strip of sub-umbrella tissue was set into pulsation by an induction shock, thus producing a single neurogenic contraction which travels through the circuit-shaped strip of tissue at a uniform rate of speed, provided that temperature, salinity, and other factors remain unchanged. It is thus possible to ascertain accurately not only the rate of nerve conduction, but also the peculiar individual characteristics of the wave in each pulsating ring. These rings were placed in a light silken bag immersed about 10 feet below the surface of the sea; and then half a stick of dynamite was exploded within 3 feet of them. This, however, produced no effect either upon their rates or the characters of their pulsation waves, although fishes possessing swim-bladders were killed within 10 feet, and injured so that they turned ventral side uppermost within 20 feet of the exploding dynamite. When the pulsating rings were placed in glass jars or tin cans, partially filled with air, the containers were crushed or shattered by the explosion, and much mechanical injury suffered by the medusa rings, which, however, could at once be restored to normal pulsation by an induction shock if their pulsations had ceased. It was also observed that the lacerated area regenerated at a normal rate. Experiments proved that fishes with swim-bladders are more sensitive to explosive shocks than those without swim-bladders: thus a half stick of dynamite, exploded within 3 feet of a small shark, which has no swim-bladder, produced no apparent injury: this applies also in a lesser degree to such teleosts as lack swim-bladders. Dr Ball dissected some of the swim-bladder fishes killed by the explosions: he found that the swim-bladder had burst, and the tissues were crushed in around it, the vertebral column being often broken. It seems, then, that in these lower forms the injurious effects of dynamite explosions, when present, are due to mechanical laceration of tissues, and especially the crushing inward of air-filled cavities. "It seems possible, therefore, that the cavities of the middle ear and Eustachian tubes may be a source of danger to men standing near exploding shells." The writer thinks that his experiments with pulsating rings of *Cassiopea* negative the suggested hypothesis that the sudden reduction in atmospheric pressure close to an exploding shell might set free dissolved gases in the blood and elsewhere, thus vacuolating the tissues and producing pressure and other effects upon the nerves; for no injurious effects other than those of simple asphyxiation were produced by sudden exhaustion of the air surrounding the

animals; and recovery, when replaced in normal sea water, was almost immediate. He holds that these results are in accord with the conclusions of Grasset, Eder, Babinski and Froment, and others, that war shock is predominantly a psychic phenomenon, and being hysteria it can be cured by hypnotic suggestion.

LEONARD J. KIDD.

NEUROSES, PSYCHO-NEUROSES, &c.

MENTAL DISTURBANCES IN SOLDIERS IN RELATION TO (277) WAR. (*I disturbi mentali nei militari in rapporto alla guerra.*)

G. SEPPILLI, *Riv. ital. di Neuropatol., Psichiatria ed Elettrotec.*, 1917, x., p. 105.

ALTHOUGH there is no special war psychosis, war favours the development of some psychopathic states rather than others, especially those in which the emotional factor and physical factor constitute an important etiological element.

Among 260 soldiers admitted to the asylum at Brescia the majority presented confusional syndromes. Other psychoses were less frequent. Neurasthenic and hysterical syndromes formed a small contingent. A few cases of epilepsy were seen, especially those showing ambulatory determinism and other atypical forms.

J. D. ROLLESTON.

WAR AND NEUROSIS. WITH SOME OBSERVATIONS OF THE (278) CANADIAN EXPEDITIONARY FORCE. CLARENCE B. FARRAR,

Amer. Journ. Insan., 1917, April, No. 4, p. 693.

AFTER a short stay in one of the base hospitals in England, Canadian soldiers who have developed mental symptoms are taken to Quebec where they are brought before a special medical board. If it appears that the patient's condition is a chronic and hopeless one, or if his symptoms are particularly aggravated, he is sent to the provincial hospital in the military district from which he enlisted. Milder cases of the shell-shock variety are sent to convalescent homes in their respective districts. All other mental cases in which special institutional treatment seems indicated are transferred to Coburg Military Hospital.

In regard to the types of case a single-diagnosis classification is held to be quite inadequate, as many of the cases are really psychiatric composites. The great majority of the cases were, however, of the dementia præcox variety, and did not vary in any essential way from corresponding civilian types.

The war neuroses fall into two groups developing (a) in camp—*anticipatory war neuroses*, (b) at the front—*trench neuroses*.

The factors leading to the development of a trench neurosis are considered under five heads:—

1. Individual psycho-physical constitution.
2. Exhaustion and kindred factors.
3. Specific motor-habit formation.
4. The psychogenic moment.
5. Trauma.

The author admits that his case material has not been large nor varied enough to enable him to draw general conclusions, but in over 90 per cent. of his cases there was a constitutional predisposition.

Exhaustion has no doubt an influence in many of the cases, but its value as a causative has been greatly over-rated.

Under specific motor-habit formation is considered what the author calls the "dodging-reflex" which is brought about by any sudden, unexpected stimulus. It would seem to the reviewer, however, that this is much more a symptom of a nervous state rather than a causative factor.

In considering the psychogenic factor two important statements are made, viz.: (1) "Among prisoners as compared with fighting troops neuroses are conspicuously rare." (2) "The absolute frequency of neurotic conditions is greater in the hospitals that are far removed from the front than in the field hospitals themselves." It is also pointed out how seldom officers as compared with men show manifestations of hysteria.

The psychogenic factor is held to be of great importance, and the war neurosis is looked upon as the embodiment of the instinctive unconscious or imperfectly conscious protest on the part of the invalid nervous system against service at the front. "It is the reactive rebellious assertion of the claims of the individual as opposed to the demands of the State."

Trauma is often the starting-point of the neurosis, but the relation between cause and effect is often not clear.

D. K. HENDERSON.

**RECOMMENDATIONS FOR THE TREATMENT OF MENTAL
(279) AND NERVOUS DISEASES IN THE UNITED STATES
ARMY.** THOMAS W. SALMON, *Psychiatric Bulletin*, 1917, July, p. 355.

THE most important recommendation to be made is that of rigidly excluding insane, feeble-minded, psychopathic, and neuro-pathic individuals from the forces which are to be sent to France. It is recommended that a special base hospital of 500 beds for the neuro-psychiatric cases be located at the base upon which army (of 500,000 to 600,000) rests. Such hospitals to be used for cases likely to recover and return on active duty within six

months; other cases to be cared for while waiting to be evacuated to the United States. In connection with this hospital there should be one or more special convalescent camps. In the advanced areas there should be special neuro-psychiatric wards of thirty beds, in charge of three psychiatrists and neurologists for each base hospital having an active service. These wards to be used for observation (including medico-legal cases) and for emergency treatment of mental and nervous cases. In the special base hospital it is specially emphasised that provision must be made for hydrotherapy, occupation, and so on. The medical personnel of such a hospital should consist of twenty officers, with suitable N.C.O.'s, nurses, and instructors for the special departments.

In the United States one or more clearing hospitals should be established for reception, emergency treatment, classification, and disposition of enlisted cases among enlisted men invalided home. Similar clearing wards should be established for officers. Legislation permitting the Surgeon-General to make contracts with public and private hospitals, maintaining satisfactory standards of treatment for the continued care of officers and men suffering from mental diseases until recommended for retirement or discharge by a special board. Appointment of a special board of three medical officers to visit all institutions in which insane officers and men are cared for under such contracts, to see that adequate treatment is being given, and to retire or discharge (with or without pension) those not likely to recover. Reconstruction and re-education centres should also be formed for cases of war neuroses (shell shock).

D. K. HENDERSON.

WAR NEUROSES. J. T. MACCURDY, *Psychiatric Bulletin*, 1917, July, (280) No. 3, p. 243.

THIS paper of 112 closely printed pages is the result of a two months' tour during which the author visited five hospitals in London, one near Liverpool, and one near Edinburgh. MacCurdy rightly objects to the term "shell shock," as it implies a single etiology—which is far from being the case; and secondly, because "the clinical types covered by this blanket diagnostic term are too various to be safely gathered under one heading."

The paper is couched in general terms: quite a large number of the author's statements are open to harsh criticism, and this cannot be wondered at, because a great many of his facts have been adduced from hearsay evidence, and it seems impossible to the writer of this criticism for anyone to give a broad survey of the clinical material, or a careful study of all the factors at work in the time which the author had at his disposal. MacCurdy has

interviewed a series of patients and has discussed the cases with other physicians. He divides the war neuroses into two main groups—(1) anxiety hysteria, and (2) conversion hysteria, and a great many exceedingly interesting case records are reported. It is not his individual cases which one would criticise but the number of wild, general statements which MacCurdy makes in an attempt to back up his own preconceived ideas in regard to war and its psychology. For instance, he makes the statement without giving his data that five officers suffer from some form of war neuroses in proportion to one non-commissioned officer or soldier. The reviewer has no definite facts to go on, but he can say that the proportion of officers to men passing through D. Block, Royal Victoria Hospital, Netley, suffering from various forms of mental disturbance, is equal. In discussing a hypothesis to account for the attitude of the average man in face of war, MacCurdy says that one of the phenomena exhibited by the war neuroses is the tendency to return to the mental attitudes of civilian life, and to become increasingly obsessed with the horror of warfare. Anyone who has seen anything of modern warfare cannot but be struck by the horror of it, but to talk about the average man having it as an obsession is not a fact. Then in discussing the average man's repugnance to war he goes a step further and says: "The doctrine of sublimation as developed by the psycho-analytic school of psychology furnishes probably the only effective explanation for the lifting of this repression in times of war." The foregoing statement is then used by the author as an explanation of how it is that the soldier can take a delight in the injuries inflicted upon his foe. There are, no doubt, certain types who do take a delight in slaughter and bloodshed, but to apply such formulæ to the average man is, it seems to me, stretching the point very considerably, and it is absurd to bring in sublimation and psycho-analysis. A terrible picture is drawn, from the author's imagination, of the effect of a bombardment,—every day the soldier grows more and more sensitive, and this "may even develop to the point of pity for the foe which is naturally most incapacitating for a soldier." Such a statement has absolutely no foundation in fact, and under a bombardment it is much more usual to hear considerable condemnation of the foe rather than pity for him. Again, on p. 263, while saying that probably there are no more fervid pacifists in existence than the men in the trenches, he adds that many of them acquire with their reluctance to bloodshed such a pity for the enemy that they find it difficult to fight effectively. In discussing those suffering from anxiety states, he says that the one so affected is always one with high ideals of duty, and therefore

such a one never entertains the hope of a disabling wound. Further on he says that a wound is looked upon as an ideal form of relief, and then follows an extraordinary sentence: "So true is this that it is said to be a common occurrence for a soldier who has had a foot or a leg blown off to dance about on the remaining one, shouting with joy that he has got a 'Blighty one.'" The above are only a few examples of the loose reasoning and ill-considered statements of the author of this paper, and if the reader can ignore them and study the cases and the points raised in connection with them he will find some useful information.

D. K. HENDERSON.

SOME NOTES ON BATTLE PSYCHO-NEUROSES. E. FRYER
(281) BALLARD, *Journ. Ment. Sci.*, 1917, lxiii., July, p. 400.

UNCONSCIOUSNESS arising from physical or atmospheric concussion due to "blowing up" or burial is, in the majority of cases, merely the last straw in the production of the psycho-neurosis. Some break down without such an incident, or if such occur, the break-down may be postponed.

Shell shock produces two main syndromes: (1) The anxiety type with tremors, perspirations, mental and somatic apprehension, insomnia, &c.; (2) hysteria, comprising delirium, stupor, automatism, amnesia (dissociations of consciousness); and somatic episodes, such as deafness, dumbness, anæsthesia, paralysis, &c. Hysterical, hysterio-epileptic, or epileptic fits may ensue; these are all hysterical in origin.

Vertigo (probably vasomotor, and of neurasthenic origin) may occur; and stammer, without anxiety symptoms. Vomiting is rare, but severe when it occurs; it is caused by emotional factors, and may be cured by psychical means.

Romberg's sign and "trombone" movements of the tongue may sometimes be seen in agitated cases.

The hypothetical ætiology of the above symptoms is as follows: Soldiers under fire, being human beings, are afraid. The instinct of self-preservation is in arms, the soldier suppresses into the sub-conscious the struggle between the instinct to run away and the necessity to "stick it out," and sublimates by allowing the fear some play in the disguised form of anger, &c. Eventually, if unable to continue suppression (failure of the censor), the fear-complex again arises, and agitated neurasthenia occurs, or even fits, if he struggles to re-suppress, and fails. On the other hand, should he be able to suppress for a prolonged period under fire without sufficient sublimation, and nothing occurs to break down the censor, then he develops eventually as an instinctive compromise some hysterical episode, *e.g.*, dissociation of consciousness,

dumbness, paralysis, &c. The author gives his theory to explain these episodes.

Severe types exhibiting well-marked symptoms after six months do not recover in the army. A considerable proportion of those who "recover" relapse later.

Therapeutically electricity, &c., is useless as compared with therapeutic conversation. Cases should be completely removed from the environment in which the illness arose with three months' leave, then to a convalescent home, and later to a command depot. In the case of a relapse such cases are of no further use to the army.

For medicine, bromide of ammonium with syr. glycerophos. co. is useful. Hypnotic suggestion cures hysterical somatic episodes, *e.g.*, dumbness; but patients exhibiting physical signs of fear as well as a somatic episode should not be treated for the latter until the former has disappeared, as otherwise agitated neurasthenia may ensue, as it does in recent shell-shock cases on removal of the episode.

H. DE M. ALEXANDER.

**RECOMMENDATIONS FOR THE OBSERVATION OF MENTAL
(282) DISORDERS INCIDENT TO THE WAR.** AUGUST HOCH,

Psychiatric Bulletin, 1917, July, p. 377.

A GUIDE to the psychiatric examination of patients with mental disorders has been drawn up with a view of obtaining a certain uniformity of observations. The guide is an adaptation of the system of examination used in the New York State Hospitals.

D. K. HENDERSON.

KORSAKOW'S PSYCHOSIS IN ASSOCIATION WITH MALARIA.
(283) HILDRED CARLILL, *Lancet*, 1917, April 28, p. 648.

THE patient was a stoker, aged 45, and was admitted to Haslar early in November 1916. He had been ill at Bombay in October 1916, and the notes said that there was then œdema of the lower limbs. On 10th December 1916 he had a proved attack of malaria. He said that he had suffered from many attacks of intermittent fever. He also had gout.

When he had recovered from his attack of malaria he presented no signs of disease in his urine, viscera, or central nervous system, with the exception that he had bilateral absence of ankle jerks. This was confirmed on several occasions. There was no other evidence of neuritis.

There was no history or evidence of alcoholic excess or of venereal disease, and the Wassermann reaction of his serum and cerebro-spinal fluid was negative. The fluid contained no cells.

His memory was very greatly impaired, and he appeared to be living over again the period of the Boer War. He thought that King Edward was on the throne, and that Lord Roberts and General Buller were in command at the battle of the Falkland Islands. He also said that the war was between England and some field force, and later that it was against the Balkan States. All his information as regards his address, his journey home from Bombay, and where he spent Christmas a fortnight before, was hopelessly confused and inaccurate. He had no recollection of being given an intravenous injection, when asked about it three hours afterwards.

AUTHOR'S ABSTRACT.

DREAMS AND THEIR INTERPRETATION, WITH SPECIAL
(284) **APPLICATION TO FREUDISM.** Sir ROBERT ARMSTRONG JONES,
Journ. Ment. Sci., 1917, lxiii., No. 261., April, p. 200.

IN the war our soldiers have shown a marked dissociation of the elements of the mind, the emotions have influenced the conduct, their attention has been engrossed and the mind has acted automatically and undirected by the will.

The author refers to the various definitions of dreams, and cites examples of dreams from historical literature.

Cognition, feeling, and will are the invariable accompaniments of every mental process, whether an object is presented from without, or its picture is experienced from within. In dreams these mental elements tend to become dissociated. The will refuses to act, and it is questionable whether a dream, once initiated, can ever be modified by the will, although some people assert that they have done so.

The recollection of the dissociated elements of a dream when recalled by the memory results in attempts to interpret their meaning. Many have endeavoured to read into them some hidden meaning, while others regard them with heedless indifference—possibly the truth in regard to dreams lies between these two extremes. When we consider the enormous number of dreams it is remarkable that there are not more than occasional coincidences.

The idea of a soul probably first arose from the effort to elicit some meaning from dream phenomena—these suggesting excursions of the soul into some distant region, which it explored, and reported what it had experienced to the waking soul. This “symbolical” view has been revived to-day, although the symbols are erroneously interpreted to be those of sexual disturbance. The “seer” of ancient times is represented to-day by the scientific psychologist.

The materials of which dreams are made are chiefly memories

of last experiences, although they are often modified by the influence of temperament and environment. Most dreams are buried in the unconscious mind—hence the difficulty in fully remembering them after waking. The age of greatest dreaming as well as that of the most vivid dreams, is from 20 to 25. Women dream more than men. The majority of dreams occur before 6 A.M. The precipitation of images in a dream is so great that its enactment in a few seconds would be equivalent to days in the waking state. Sixty per cent. of dreams relate to sight; 5 per cent. to hearing; 3 per cent. to taste; and 15 per cent. to smell.

The mental faculties "go to sleep" in certain orders—the power of judgment and association being first lessened.

Dreams may be followed by insanity, though it is doubtful if they can cause insanity except in the predisposed. The insane dream more than the sane, and on account of their ever present hallucinations are on the alert and are therefore light sleepers. The dreams of many wounded soldiers are associated with the physical signs of fear.

Somnambulism, reverie, "day-dreaming," "trance," "lethargy," "catalepsy," and the "hypnotic" state are also related to the dream state.

It is preferable to limit the term "subconscious" to the state in which thoughts are present in the field of consciousness but are unnoticed owing to inattention, while the "unconscious" state represents the unconscious area into which thoughts emerge and which could not attract attention until their position had been raised into the clear focus of attention by some association or suggestion.

The psycho-analyst regards dreams as the resultant of a conflict between the censor and the repressed idea, and he regards the unconscious mind as an under-world of painful memories kept in subjugation by the "censor." This is not in accord with experience, as we cannot forget unpleasant scenes or thoughts. The Freudians, who urge sex as the basic origin of all dreams, &c., are "sex-intoxicated" and read into dreams the fantasies of their own auto-suggestions.

Insane criminals dream much as other people; the discovery of crime through the criminal giving himself away in dreaming is unknown to the author, and there is no record of psycho-analysis assisting in the detection of crime.

We dream most of events which have received no attention during the day; were it otherwise the necessary restoration and nutrition of the brain during sleep would be impossible.

H. DE M. ALEXANDER.

MENINGITIS.**THE PRE-MENINGITIC RASH OF CEREBRO-SPINAL FEVER.**

(285) C. P. SYMONDS, *Lancet*, 1917, ii., July 20, p. 87.

THREE cases are described in soldiers. The first case was under observation from the onset, and showed a profuse rash six hours later, which disappeared four hours after it had been first observed. The rash in the second case was erythematous, and thought at one time to be due to measles. The spots were raised, and averaged about the size of a threepenny-piece. In the third case the rash was present twelve hours after the onset, and had almost disappeared six hours later.

The author thinks these three cases tend to confirm the view that there is a septicæmia stage in cerebro-spinal fever which precedes the meningitis.

A. NINIAN BRUCE.

INCOMPLETE CEREBRO-SPINAL MENINGITIS. (Meningite

(286) *cérébro-spinale fruste*.) H. RENON, *Paris méd.*, 1917, vii., p. 542.

DURING an epidemic of cerebro-spinal meningitis a soldier, aged 30, who had recently undergone anti-typhoid inoculation, fell ill, with vomiting, headache, and fever. There was no nuchal rigidity or Kernig's sign. An eruption of facial herpes, however, and a slow pulse (40) suggested meningitis. On lumbar puncture the cerebro-spinal fluid was clear, but under slight hypertension, and contained meningococci. After the puncture the pulse rates became normal. Renon thinks that typhoid inoculation rendered the organism more liable to infection, and should therefore be avoided in an area where an epidemic of any kind is prevalent.

J. D. ROLLESTON.

CHEMICAL TREATMENT OF MENINGITIS. (Sur la traitement

(287) *chimique des méningites*.) CARLOS FRANÇA, *Compt. Rend. Soc. de Biol.*, 1917, lxxx., 5 Mai, p. 422.

IN 1902 França introduced the treatment of non-tuberculous meningitis by intraspinal injections of lysol solution. His method has been found to be very effectual in severe cases: the injections are well borne, and no bad effects follow. In cases of epidemic cerebro-spinal meningitis the lysol acts as an antiseptic agent, and the diplococci quickly disappear from the spinal fluid. The method he uses is as follows: After removal of 25 to 50 c.c. of spinal fluid by lumbar puncture he injects intraspinally a 1 in 100 solution of lysol; the amount varies according to the patient's age, 12 to 20 c.c. for adults, 3 to 9 c.c. for children. If the patient's state be

very grave, daily injections are given until the spinal fluid becomes sterile: this usually occurs rapidly. The only sequela observed is a yellowish tint of the palms and soles. In cases of purulent meningitis the lysol injection is preceded by lavage with normal saline solution. After the lysol injections the patient is put into an inclined position, with lowered head. Urotropine is given as an adjuvant. França advises that his method should be used in cases of epidemic cerebro-spinal meningitis when, in spite of serum treatment, the course of the meningitis is slow and the meningococcus persists in the spinal fluid; and in all bacterial forms of meningitis, with the exception of the pneumococcic and the tuberculous.

LEONARD J. KIDD.

THE RÔLE OF THE PHAGOCYTE IN CEREBRO-SPINAL MENINGITIS. CRESSWELL SHEARER and H. WARREN CROWE, *Proc. Roy. Soc.*, 1917, lxxxix., Series B., pp. 422-439.

EXPERIMENTS are described demonstrating that under certain conditions the meningococcus can be taken up by the leucocytes but not killed by them. In the case of freshly isolated strains the leucocytes will not take them up at first, while old laboratory cultures are ingested with great rapidity. This happens also with most of the nasal strains examined from chronic "carriers."

In the intermediate stage between the fresh spinal condition and the naso-pharyngeal state it can be shown experimentally that they are taken up, but not killed, by the leucocytes. They can be removed from them after a period of twenty-four, forty-eight, or even sixty hours and grown on artificial media. If we can believe that they behave similarly within the body, then we can understand how they might be carried into the spinal canal and there set up infection. It might also explain why direct infection (apart from the "carrier") seldom if ever takes place in cerebro-spinal fever, *i.e.*, from one patient to another, because the phagocytes refuse to take up the germs in their virulent condition. In the "carrier," on the other hand, the germs have lost their virulence so completely that they are taken up and immediately killed and digested.

The virulent organism being thus unsusceptible to attack by the phagocytes, and the longer the germs grow in the "carrier" throat, the more easily will they be ingested until a time is reached when, on ingestion, they are also destroyed. Somewhere between these extremes infection may produce the disease. The organism is sufficiently weak to give in to the leucocyte attack, but not to lose its life in the battle. Should infection occur at this point, the leucocytes will pick them up from the mucous membrane of the naso-pharynx, and in the course of their

wanderings will sometimes carry them into the spinal canal. There the liberated organisms will set up the disease, at the same time re-acquiring the power of resisting the attacks of the leucocytes in the presence of normal serum.

A. NINIAN BRUCE.

THE PURPURIC FORMS OF CEREBRO-SPINAL MENINGITIS.

(289) (*Les formes purpuriques de la méningite cérébro-spinale.*) A. NETTER, *Rev. de méd.*, 1916, xxxv., p. 133.

CEREBRO-SPINAL meningitis may be accompanied by purpura, which may precede the meningitis or occur without any meningitis at all.

The purpura indicates that the infection is becoming generalised, and is assuming a septicæmic character. The proof of this is furnished by the demonstration of the meningococcus in the skin lesions and the blood, and by the coexistence of other extra-meningeal lesions (irido-cyclitis, arthritis, endocarditis, &c.).

These purpuric manifestations have been decidedly more frequent during the last two years in England and Germany, as well as in Paris. They have also been infinitely commoner in other countries in past times, *e.g.*, North America in the beginning of the nineteenth century, and in Ireland in 1866.

The diagnosis of these lesions may be very difficult, especially when the signs of meningitis are absent, or when there is no meningitis at all. The special gravity of purpuric meningitis demands an early serum treatment. The intravenous method which involves certain dangers is not required in every case. It will be best even in cases of meningococcal infection without meningitis to begin with intrathecal injections.

Polyvalent serums should be employed which can deal with the largest possible number of different strains of meningococci. "It is doubtless due to the absence of this property that the failure and scepticism of our English confrères is due who record a mortality of over 60 per cent." (*v. Revue*, 1916, xiv., p. 36) "with serum treatment, whereas in our country the total mortality is 24 per cent."

J. D. ROLLESTON.

ACUTE SYPHILITIC MENINGITIS. S. A. K. WILSON and A. C. E. (290) GRAY, *Brit. Med. Journ.*, 1917, ii., p. 419.

MOST of the cases of acute syphilitic meningitis on record have been reported by French writers, whose papers have been abstracted in this *Review*, *e.g.*, Boidin and Weill, *Review*, 1907, v., p. 901; Ravaut and Darré, *ibid.*, 1907, v., p. 718; Laubry and Giroux, *ibid.*, 1908, vi., p. 174; Claisse and Joltrain, *ibid.*, 1908, vi., p. 290; Ballet and Barbé, *ibid.*, 1908, vi., p. 419; De Coux, *ibid.*, 1908, vi., p. 599; Achard and Desbouis, *ibid.*, 1913, xi., p. 38; Jeanselme,

ibid., 1913, xi., p. 39; Bronstein, *ibid.*, 1913, xi., p. 167; Audry and Lavau, *ibid.*, 1914, xii., p. 213; and Lavau, 1914, xii., p. 493.

The present case is that of a soldier, aged 24, who contracted syphilis in August 1916, and was given seven intravenous injections of salvarsan. The Wassermann reaction was positive in the blood, and apparently no secondaries developed. In November, almost exactly three months after injection, he suddenly began to suffer severe headaches, and in a few days developed characteristic symptoms of acute meningitis. The case was diagnosed as cerebro-spinal fever, and treated with full doses of anti-meningococcal serum for a week. Doubts were then cast on the diagnosis owing to the course of the disease, and the cytological findings as well as the history. Syphilitic meningitis was diagnosed. The Wassermann reaction was found positive in the blood and cerebro-spinal fluid, and after energetic treatment with mercurialised serum and mercurial injection, recovery took place four months after admission to hospital.

J. D. ROLLESTON.

TETANUS.

LOCALISED TETANUS. (*Contribution à l'étude des tétanos localisés.*) (291) ANNE ORHAN, *Thèses de Paris*, 1916-17, No. 113.

THE thesis contains the histories of eight cases, all but two of which are original, in which the tetanus was confined to one or both of the upper or lower limbs. All recovered. Five occurred in wounded soldiers who had already been given a single dose of tetanus antitoxin at the time of their wound. The writer attributes the localisation of tetanus to incomplete preventive serotherapy, and to bacilli of a low degree of virulence.

J. D. ROLLESTON.

TETANUS, WITH SECONDARY MULTIPLE NEURITIS. REPORT (292) **OF A CASE WITH RECOVERY.** W. W. RICHARDSON, *Journ. Amer. Med. Assoc.*, 1917, lxxviii., June 2, p. 1611.

AN Italian, aged 19, developed tetanus about the tenth day, following a slight lacerated wound of the finger which had not been cared for properly. Under treatment by antitoxin, magnesium sulphate, and phenol he showed two periods of temporary improvement, finally on the twenty-fifth day ceased to have convulsions, and developed neuritic symptoms in the arms three weeks later; this rapidly developed into general peripheral neuritis. After two weeks he began to improve, and by the fourth week of his neuritis had regained the power of walking a little. Approximately 150,000 units of antitoxin were used, divided nearly equally among the subcutaneous, intravenous, and intraspinal routes. The most probable cause of the neuritis was the tetanotoxin.

LEONARD J. KIDD.

GENERAL

NEURASTHENIA IN SOLDIERS OF THE HOME FORCES. F. W. (293) BURTON FANNING, *Lancet*, cxcii., 1917, June, p. 907.

THE author uses the term neurasthenia in its widest sense to include diseases of the nervous system which are believed to have no organic basis. He was astounded at the prevalence of neurasthenia in the occupants of beds in a military hospital with the consequent loss of man power, the expense, and the ensuing moral and physical deterioration of the patients. To a large extent these losses could be prevented.

Out of 1,600 home forces cases admitted to a military hospital, 509 were suffering from neurasthenia, and free from any organic disease. Of these a large number realised their nervousness as their disability, a larger number admitted their nervousness, but did not connect it with their supposed malady; the remainder believed they had disease of some organ, and denied nervousness; and, with few exceptions, all believed in their unfitness for service. All had the name of some disease ready on their tongues.

A natural lack of constitutional vigour was characteristic; and some were degenerates.

The author details the early life typical of a neurasthenic: the faulty upbringing, the introspective habits, the dislike of sport, and later, their sedentary occupation. Few enlisted voluntarily, and with the majority compulsory service started them on soldiering with the conviction that their strength would not enable them to stand the hardships of the service. Whatever finally determined their breakdown they had almost invariably suffered in a similar manner previously.

Symptoms and Signs.—The first point in the diagnosis of neurasthenia is the widespread distribution of the symptoms. Weakness, prostration, a feeling of exhaustion or sudden collapse, tremors of upper lip, closed eyelids, and extended fingers, cold extremities, hot and cold flushes, profuse sweating, loss of weight, exaggerated knee jerks, a tongue giving the appearance of having been dried by a cloth and thinly sanded or coated (many constantly moisten their lips on account of this). In more than half the patients the fields of vision were normal, and few had anaesthesia. Insomnia was not complained of. The symptoms were worse in the morning.

Chief Complaints.—In order of frequency rheumatism comes first with pains in legs, back, &c., with normal joints and no real limitation of movement (when shown that his joints are free and

assured that only a nervous condition is present it is easy to get these men out of bed). Their claim to having had rheumatic fever in the past did not stand cross-examination. If their pains were called neuritic no signs of such were present (careful measurements here exclude atrophy). Disquietude of mind is the most common cause of headache and backache.

Cases alleged to be gastritis on account of pain brought on by food, external tenderness, vomiting (in small quantities and jealously preserved), flatulence, &c., are all probably nervous in origin. Heart pain is generally well below the heart. Palpitation, dyspnœa, giddiness (labelled "Soldier's Heart") are almost constantly present. Many had a pulse rate of 100 during waking hours which disappeared in sleep, as well as increased frequency of respiration—over-excitability of the cardiac and respiratory nervous centres. The giddiness and fainting are more often due to emotion than exercise. Many were non-smokers.

In alcoholic cases (where remorse plays a part) there is always the probability of some organic change. In only two cases was an enlarged thyroid present, and here there was more interference with the general health, and the quickened pulse did not disappear during sleep.

Tuberculosis of the lungs, bladder troubles, throat affections, have all been suggested by the symptoms present. The sexual organs were less frequently involved than in the civilian neurasthenic.

Prevention.—A great deal of neurasthenia is due to an incorrect diagnosis and the failure to disabuse the patient's mind of his apprehensions about the presence of organic disease. Its prevention comes within the sphere of education—the atmosphere of our public schools prevents it. All classes are affected; but particularly that class whose education has been carried a little further than that of their station, but not yet far enough. There would be fewer breakdowns if recruits were better classified and gradually got on to full drill. Self-confidence must be established in these men.

Treatment—Prognosis.—A neurasthenic will not begin to get well until he is persuaded he is free from any actual disease. Make a careful physical examination and gain his confidence. The mere unburdening of his soul confers relief on the patient. Occupation is as a rule better than rest. Few of these men can be made into real soldiers, but after treatment they might be made efficient workers behind the line or at home.

H. DE M. ALEXANDER.

**A CASE OF HYPERTROPHIC MYOPATHY OF THE UPPER LIMB,
(294) WITH PARALLEL HYPERTROPHY OF THE SKELETON.**

(Un cas de myopathie hypertrophique du membre supérieur avec hypertrophie parallèle du squelette.) H. CLAUDE and J. LIHERMITTE, *Paris méd.*, 1917, ii., p. 49.

THE patient was a soldier, aged 23, who presented hypertrophy of all the tissues of the left upper limb and adjacent part of the thorax. The condition had first appeared at the age of 10 years, and had considerably increased since he had been wounded in the shoulder, when the musculo-spiral was slightly involved.

The hypertrophied muscles, especially those of the hand, the biceps, and deltoid, showed constant myoclonic contractions. Their consistence and electrical reactions were normal. In spite of their increase in size, the hypertrophied muscles were considerably weaker than those on the opposite side. Biopsy showed an absence of any gross lesions, but in some of the fibres there was found to be a penetration of the nuclei of the sarcolemma into the actual substance of the muscle fibre. J. D. ROLLESTON.

TOLERANCE FOR ADRENALIN IN ADDISON'S DISEASE.

(295) P. NOLF and H. FREDERICQ; *Arch. Méd. belges*, 1917, lxx., p. 691.

A SOLDIER, aged 38, presenting the principal symptoms of Addison's disease, was treated by subcutaneous and intravenous injections of adrenalin, being given 10.5 mgm. in four and a half hours (2 mgm. subcutaneously and 8.5 mgm. intravenously). The blood pressure, however, was never restored to a satisfactory level, nor did glucose appear in the urine, whereas a normal individual almost always presents glycosuria after a subcutaneous injection of 1 mgm. of adrenalin. No bad effects were noted from the intravenous injection of the drug. J. D. ROLLESTON.

TUMOUR OF THE CORPUS CALLOSUM. ANTHONY HARRIGAN and

(296) JOSEPH BYRNE, *New York Acad. Med.* (Sect. Surgery), Oct. 5, 1917 (*Med. Record*, 1917, xcii., Oct. 27, p. 742).

PATIENT was a man of 47, a crucible steel worker. *General Symptoms*:—Headache, choked disc, vomiting, irritability, failure of memory and of concentration. *Local Symptoms*:—Right hemiparesis, twitching of right hand, general slight impairment of sensibility (pressure pain, posture, and passive movements), more marked on right. Astereognosis, especially on right; smell and taste impaired on left, and hallucinations of taste. Blood, Wassermann, and X-ray examinations negative. Diagnosis was sub-cortical lesion under left Rolandic region, corresponding to the right-hand area. Operation advised and performed: an

osteoplastic flap turned down over the Rolandic region revealed a plaque of pachymeningitis. No tumour or cyst was visible or palpable. Patient never regained consciousness, developed œdema and coma, and died sixteen hours after operation. Necropsy shows after death: the brain was sectioned vertically by Dr John H. Larkin, and the sections studied serially. He found a tumour, 8×6 cm., in the white matter of left hemisphere, extending from beneath the Rolandic cortex down into the corpus callosum, and backwards into the occipital lobe. The microscopical diagnosis was infiltrating mixed cell sarcoma. "The significant points in the case were: (1) the heredity; parents, brothers, sisters died young; a history of headaches and insanity in mother, and of tuberculosis in three brothers; (2) the repeated injuries of head; and the convulsions, hysterical in type, appeared years after the injuries. It was possible that the first and second of these injuries might have played a part in the development of the neoplasm; (3) hallucinations of taste, possibly from indirect involvement (pressure) of the uncus and hippocampal regions on the left; (4) twitching and, later, paralysis of right hand, which were the chief localising signs; (5) the alternating slow and rapid pulse and the Cheyne-Stokes respiration, both of which fitted well with the increased pressure found at operation. The comparatively low blood pressure represented probably vasomotor exhaustion; (6) in a similar case a primary decompression would probably best meet the indications, leaving more radical measures for a subsequent occasion." (It is not mentioned whether apraxia was present or not.)

LEONARD J. KIDD.

RENFREW DISTRICT ASYLUM AS A WAR HOSPITAL FOR
 (297) **MENTAL INVALIDS: SOME CONTRASTS IN ADMINIS-**
TRATION. WITH AN ANALYSIS OF CASES ADMITTED
DURING THE FIRST YEAR. R. D. HOTCHKIS, *Journ. Ment.*
Sci., 1917, April, p. 238.

THE administrative changes necessitated by the conversion of an asylum into a war hospital are described in the first part of this paper.

The second part deals with the patients: of these 111 were non-expeditionary, and 831 had served in one or other of the expeditionary forces. The paper deals with the latter class alone, which included five German prisoners. The analysis of these was as follows:—

Manic-depressive.—188 cases (21 per cent.). Mania, melancholia, and stupor. Resembled civilian cases in their symptoms modified by the experiences they had passed through. A great many had had previous attacks.

Alcoholic Insanity.—152 cases (18 per cent.). The delirium tremens cases exhibited two types—one who broke down as soon as the alcohol was cut off on going on board the leave boat, the other showed no signs till again in the firing line. In contrast to these were the chronic delusional cases—generally elderly men stationed at the base. Another class exhibited various symptoms; confusion, depression, excitement, and generally always hallucinations. The campaign precipitated the mental breakdown in all these alcoholic cases and 18 of them were cases of cut throat.

Mental Deficiency.—151 cases (18 per cent.). Included all degrees of weak-mindedness, and divisible into those who were vicious or moral imbeciles (37—mostly habitual criminals) and ordinary defectives—these latter proved useless and often dangerous (loading their rifles without orders) to their comrades. Some of these cases exhibited confusion, acute excitement or depression from which they recovered.

Confusional Insanity.—134 cases (16 per cent.). Twenty-seven exhibited acute symptoms. Many were irritable, had tremors and hysterical symptoms. The majority of these cases occurred on active service from mental or physical strain and their appearance suggested a neurotic temperament.

Dementia Præcox.—118 cases (14 per cent.). Of these 93 were simple, 11 catatonic, and 14 paranoid. (Many of the manic-depressive cases may ultimately prove to be really cases of this affection.) Some of these cases broke down before they had reached the firing line. It is impossible to say whether the patients in this group would have carried on in civil life without a breakdown.

Paranoia.—44 cases (5 per cent.). Here the delusions had in many cases been present before enlistment and the campaign accentuated them.

General Paralysis.—22 cases (2 per cent.). The ordinary symptoms were present. In a few of these cases the progress of the disease was not materially affected by the campaign.

Other Organic Brain Conditions.—These were single cases of tuberculous meningitis, hemiplegia, destruction of brain tissue from a kick from a mule, cerebral abscess secondary to a gunshot wound, and syphilitic meningitis.

Few cases of head injury exhibited mental symptoms.

Epilepsy.—7 cases. All except one had epilepsy prior to enlisting.

Secondary Dementia.—7 cases. All transfers.

Not Insane.—4 cases. One, a malingerer with aphonia, was anæsthetised three times with ether and wakened when shouting, but resumed silence on realising that he was speaking. He admitted recovery prior to the fourth anæsthetation.

Mental Instability.—A useful term to apply to recovered cases who on account of previous attacks should return to civil life.

500 expeditionary cases were discharged. Of these 155 returned to duty, 139 were sent to asylums, 40 recovered, and 111 were removed, relieved, by their friends. Forty-two were removed to other hospitals for treatment of their bodily condition; 11 died, and 2 escaped.

H. DE M. ALEXANDER.

Reviews

HYSTERIE-PITHATISME ET TROUBLES NERVEUX D'ORDRE
(298) **RÉFLEXE EN NEUROLOGIE DE GUERRE.** J. BABINSKI and
FROMENT. Illustr. Collection Horizon. Masson et Cie, Paris. 1917.
Pr. Fr. 4.

IN the first part of this most interesting book Babinski and Froment review the results of the former's investigations on hysteria made since 1896 in the light of their observations on soldiers during the present war. In the second part they describe a number of nervous disorders which are neither truly organic nor hysterical, but are in their opinion reflex in origin. Babinski's views on hysteria have gradually gained acceptance in France, and in the discussion on hysteria at the Société de Neurologie in 1908 every speaker was fundamentally in agreement with him. For this reason he regards the second part of the book—on reflex disorders—as the more important, as it contains a large number of completely new observations; but for English readers the first part is of equal, if not of greater value, as Babinski's remarkable and convincing researches on hysteria are still to a great extent unknown or misunderstood by British physicians.

According to Babinski, hysteria is a pathological condition manifesting itself by symptoms which can be exactly reproduced by suggestion in certain individuals, and which can be caused to disappear under the sole influence of persuasion and contra-suggestion.

Others had taught the importance of suggestion in the production of hysterical symptoms, but it was generally believed that emotional disturbances could also give rise to them without the intervention of suggestion. The authors discuss the subject from their own experience in peace and war, and from the accounts given by trained observers, who themselves happened to be on the scene at times of great emotional stress, such as earthquakes, railway accidents and shipwrecks, and heavy bom-

bardments at the front. They quote Clunet's remarkable account of the nervous phenomena he observed in February 1916, when the transport "La Provence," on which he was travelling, was torpedoed. They conclude that emotions never give rise automatically and immediately to hysterical symptoms, as they do to tachycardia or sweating. There is always an interval before they appear—Charcot's "phase of meditation"—during which auto- and hetero-suggestion can act. Thus Clunet saw no hysterical symptoms during the seventeen minutes which elapsed after "La Provence" was torpedoed and before she sank, nor during the eighteen hours he spent on a raft. There were manifestations of extreme emotion in the boat, and of physical suffering on the raft, but it was not until they were picked up by a destroyer that numerous men showed hysterical symptoms, all of which Clunet cured within a few hours by vigorous contra-suggestion, with such success that no relapse occurred during the eight following days whilst they remained under observation. Corresponding with this, hysterical manifestations are very rare in the trenches, though emotional symptoms are common enough, many men reaching the regimental aid posts weeping and tremulous, with rapid pulse and respiration. In this emotional state they are always more or less confused, the confusion being associated with an abnormal degree of suggestibility, weakening of the critical faculties, and absence of initiation. It is in the clearing stations, at a distance of ten miles or more from the front, that hysterical symptoms first become manifest, and they are still more common at the base. Here, as the dazed soldier begins to recover from his confusion, he tends to exaggerate and perpetuate the difficulty he experiences in summoning up the energy required to perform the various functions of his body, and instead of speaking or walking, he suggests to himself that he is dumb or paralysed. So far from hysterical symptoms being produced by emotions, the latter, if sufficiently powerful, are very likely to cure them.

Thirty years ago Charcot showed how many of the symptoms resulting from accidents, which had previously been regarded as organic, were really hysterical in origin. But injuries, like emotions, do not themselves produce hysterical symptoms. The war has confirmed the conclusions reached from experience gained in civil life that injuries, including the concussion of shell shock, still further exaggerate the abnormal suggestibility which results from the emotions caused by the events preceding and accompanying the injury. The direct physical results of the injury often suggest the particular form the hysterical manifestation should take, the symptoms being of a nature which the patient himself would expect to result from his wound.

Hysterical symptoms have been common enough during the war, but in France, as in England, many nervous conditions have been described as hysterical, although they were not due to suggestion nor curable by contra-suggestion, and were therefore not really hysterical at all. The distinction is of great importance, as the prognosis, treatment, and military disposal of hysterical cases differ from those of all other conditions. The authors describe the various hysterical symptoms and their differential diagnosis in detail, but only those in which their views are of special interest need be mentioned in this review.

Charcot taught that hysteria manifests itself in two ways—one by permanent signs or stigmata, of which the patient is unaware, and the other by a great variety of symptoms, of which the patient is aware, and for the cure of which he seeks medical aid. Among the so-called hysterical stigmata are pharyngeal anæsthesia, hemianæsthesia, unilateral diminution in the acuity of the special senses, and concentric retraction of the field of vision. Babinski and Froment, in common with the majority of French neurologists, have failed to find any evidence among hysterical soldiers of the existence of these stigmata. On the other hand, they are incidentally described in several papers on war neuroses by British writers. My own experience entirely supports Babinski's view that the stigmata are invariably produced by suggestion, generally by the observer himself. They are not permanent symptoms indicating the hysterical soil on which the new hysterical symptoms have grown, but like the latter they have been produced by suggestion, and can be made to disappear by suggestion. Their presence cannot be taken as evidence that the symptoms which accompany them are hysterical, as individuals suffering from certain organic lesions and functional conditions other than hysterical, especially soldiers who have spent many months at the front, are more suggestible than the average man, so that a careless examination may produce every stigma which is looked for. The doctrine of hysterical stigmata is most misleading, as their discovery may cause organic symptoms to be regarded as hysterical, and the organic element may be missed in conditions in which organic and hysterical symptoms are associated together. Conversely, hysterical symptoms, which have developed as a result of very powerful suggestion in an individual who is not more suggestible than an average healthy man, as not infrequently happens in soldiers, may be regarded as organic, because the methods used in looking for stigmata do not produce a sufficiently strong suggestion to cause them to appear. The nervous symptoms regarded as stigmata of hysteria vary in frequency according to the observer. They are very frequently found by those who have been taught

to look for them as genuine signs of hysteria, but they are never found by those who know that they only occur as a result of the physician's suggestion. They are of the same nature as the transitory hysterical symptoms, being produced by suggestion, and being easily removed by suggestion, but they are produced by hetero-suggestion instead of by auto-suggestion.

Retraction of the field of vision has long been regarded as the most characteristic "stigma" of hysteria. Janet considered it to be "the emblem of hysterical sensibility in general," and it led him to describe hysteria as a condition due to retraction of the field of consciousness. Babinski and Froment quote the opinion of the distinguished French oculist, Morax, a former assistant of Charcot, from whom he learnt the supposed significance of the retracted field of vision. Although at first he continued to find it in almost every patient suffering from hysterical symptoms, his experience in the war has confirmed the opinion he has now shared with Babinski for several years—that it never occurs unless it is produced by suggestion on the part of the observer, and that it is therefore not a stigma at all. No patient ever spontaneously complains of it. In using a perimeter on a highly suggestible patient suffering from hysterical symptoms, it is exceedingly difficult to avoid suggesting a narrow field of vision, the mere explanation of what is going to be done being often sufficient. Since recognising the fallacious methods he had used for testing the fields of vision, Morax has substituted others, in which the finger or some other familiar object is used. At first they seem less accurate, but they are in fact much more accurate, as suggestion can be more easily avoided.

Hysterical attacks have been common both in French and English soldiers. They can be distinguished from true epilepsy by the objective signs of the latter, such as cyanosis of the face and an extensor plantar reflex. The other symptoms of an epileptic fit—the initial cry and sudden onset, complete loss of consciousness, bitten tongue, blood-stained froth on the lips, micturition, and subsequent depression are important, but not conclusive, as they can be imitated consciously by the malingerer or unconsciously in hysteria. Hysterical attacks are often easy to recognise, when they show the gross movements, gesticulations, and *arc de cercle*, but conclusive evidence in many cases can only be obtained by reproducing the fits by suggestion. This is particularly valuable when no fit has been witnessed. Babinski used faradism as a means of suggesting a fit; I have suggested fits with equal success under hypnosis. In both cases they can be instantly terminated by a further suggestion. It is necessary to remember that hysterical and epileptic fits may occur in the same individual. I have seen

three undoubted cases of this in soldiers, the hysterical fits being cured by suggestion under hypnosis, and the epileptic fits controlled by bromide.

No patient ever seeks medical advice on account of hysterical anæsthesia. The authors quote Lasèque, who found that no patient ever mentioned anæsthesia, although asked to describe his symptoms in the greatest detail, but the moment it has been produced by suggestion he may complain of the inconvenience he suffers from it. The condition never leads—like the anæsthesia due to organic lesions—to burns or other injuries. If great care is taken to avoid suggestion whilst examining patients with hysterical symptoms, no anæsthesia is ever found if the sensory functions have not already been investigated. This was Babinski's experience in over 100 patients suffering from hysterical symptoms whom he examined with extreme care, and it is confirmed by numerous authors in France, and with two possible exceptions by my own experience, although until I first visited Babinski's clinic in 1907 I had believed that anæsthesia was a common hysterical symptom, and had found it very frequently in the well-known forms such as hemi-anæsthesia. The anæsthesia may vary from day to day according to the conscious or unconscious suggestion of the observer, and tends to disappear spontaneously if ignored. Babinski never allows the patient to compare one side with the other—a method which always suggests to him that some difference is probably present. He examines all the various superficial and deep sensations over all parts of the body indiscriminately without any order, the patient simply stating what he feels, so that it is almost impossible for the patient to develop a definite area of anæsthesia to some or all stimuli as a result of suggestion. The anæsthetic areas which are often considered as characteristic of hysteria are simply the areas of anæsthesia which are likely to result from a careless examination, in which sufficient care has not been taken to avoid suggestion. Moreover, hemi-anæsthesia may occur in organic disease, and areas on the limbs like those supposed to be characteristic of hysteria have been observed in medullary and spinal hæmorrhage and in syringomyelia.

The authors have found that hysterical contractures are much less common in soldiers than was at first believed, as contractures which they regard as reflex in origin have often been erroneously diagnosed as hysterical. Under an anæsthetic the former persist until the patient is deeply anæsthetised, and the deep reflexes are then found to be increased, whereas in hysteria they relax at once, as they do in sleep, and the reflexes are unaltered.

The war has brought no new facts to light which alter Babinski's opinion that the cutaneous reflexes are never abolished or unequal

in hysteria, even when hysterical anaesthesia is present owing to suggestion at a previous examination. Occasionally the plantar reflex appears to be abolished, but in such cases the muscles of the leg, foot, and especially the toes, are not relaxed, corresponding with the fact that by an effort of will it is possible to mask the reflex by keeping the limb rigid. If sufficient relaxation can be induced, the reflex can be obtained. In other cases the absence of the reflex is due to the foot being cold; the reflex then appears on warming the foot.

In numerous papers published since 1893, Babinski has proved that the tendon reflexes are unaltered in hysteria. They are never unequal in hysterical hemiplegia, abolished in flaccid paraplegia, or exaggerated in pseudo-spastic paraplegia. Most apparent exceptions have proved to be due to errors in diagnosis, the presence of organic disease being ultimately proved. The remainder have been due to ignorance of the variations which are possible in normal individuals, as the knee jerks may occasionally be very difficult to obtain or extremely brisk in the absence of any organic or functional disease. The pseudo-ankle clonus, sometimes seen in hysterical paralysis, is due to absence of muscular relaxation, and can be obtained in a normal individual if the proper degree of muscular rigidity is assumed.

The authors find that the experience of the war confirms the belief they previously held that œdema, vasomotor disorders, and trophic lesions are never hysterical in nature. Though cases of hysterical subcutaneous œdema, cutaneous erythema, bullæ, ulceration and gangrene, visceral hæmorrhage, pyrexia, and tachycardia were at one time frequently described, no authentic case has been reported in France since the Paris discussion on hysteria in 1908. The change in opinion is due to improved diagnosis and a more accurate knowledge of the nature of hysteria. So-called hysterical œdema was due to some organic vascular or nervous lesion, which had escaped recognition, or was produced by a malingerer by purposely constricting a limb. Vasomotor disturbances supposed to be hysterical are really due to organic lesions, with the exception of the slightly deficient circulation and consequent coldness which may be present in an hysterically paralysed limb owing to the absence of normal movements depriving the peripheral circulation of an important stimulus. It is now universally acknowledged that cutaneous ulceration and gangrene occurring in hysterical individuals are purposely produced by caustics. When internal hæmorrhage and pyrexia occur in nervous individuals in the absence of obvious organic disease, they are not hysterical, but are either the result of fraud or of organic lesions, which give rise to no other physical signs.

Vasomotor instability, hypersecretion of sweat, diarrhoea, and tachycardia are common in soldiers suffering from war neuroses, but they are not hysterical. They are the direct result of emotions and not of suggestion, as they can only be produced indirectly by suggesting an emotion, and their form, intensity, and duration cannot be determined by suggestion. Lastly, dermatographism, which is sometimes regarded as an hysterical symptom, can be produced as frequently in normal individuals as in individuals suffering from hysterical symptoms.

It was formerly thought that while some hysterical symptoms, like *grande hystérie*, the form of hemiplegia in which the paralysed leg is dragged behind like an inanimate object, astasia-abasia, and mutism, could be recognised by their gross characteristics, others could simulate organic symptoms so closely that the only method of diagnosis was the discovery of hysterical stigmata. As the latter have now been shown to be of no importance, reliance must be placed upon the various physical signs of organic disease, which have been discovered during the last twenty years, as these are never present in hysteria, however closely the symptoms may appear at first sight to resemble an organic condition. An excellent description is given of all these signs, many of which we owe to Babinski, in addition to the familiar extensor plantar reflex, which is known after him, and the combined flexion of the thigh and pelvis, which I have described for some years as "Babinski's second sign."

In the early stages, as for instance at the clearing stations where many hysterical symptoms are first recognised, a rapid, complete, and permanent cure almost invariably results from active contra-suggestion. The later treatment is undertaken the more difficult it is, but few cases resist contra-suggestion, except occasionally when complicated by mental symptoms, or when the treatment meets with active opposition by a patient who does not want to get well.

It is difficult to summarise the important observations made by Babinski and Froment on the reflex neuroses. Readers of their book will obtain a satisfactory explanation of many cases, the nature of which has hitherto been obscure to them. Reflex paralysis and spasm of muscles, which may occur in a limb which has been wounded, or injured by frost, or in other ways, may be present together in the same or different segments of a limb. They are accompanied by muscular atrophy, exaggeration of tendon reflexes and hypotonus, increased excitability and slow contraction of muscles on mechanical stimulation, and increased or diminished electrical excitability without reaction of degeneration occur, together with exaggeration of mechanical and sometimes

of electrical excitability of nerves. Excessive wasting and diminution or loss of sensation are present over the affected segment, and trophic changes in bones and skin may occur. When the surrounding temperature is low the cutaneous reflexes are lost, the affected part becomes painful, cyanosed, and cold, and the maximum oscillation of the pulse, as measured when the blood pressure is estimated, is reduced.

These reflex symptoms resemble hysterical symptoms in being quite out of proportion with the often trivial injury which gives rise to them, as they extend above as well as below the injury in areas corresponding with no special anatomical distribution. But they are entirely unaffected by suggestion, the tendon reflexes are often exaggerated, and the muscles and sometimes the nerves are abnormally irritable to mechanical stimulation, the response being unusually prolonged. Babinski and Froment have shown that in some cases these signs are only obvious when the patient is given a general anæsthetic; at the same time any contracture which is present relaxes less readily than in hysteria. The position assumed by the limb does not necessarily correspond to any position which could be assumed voluntarily, as is always the case in hysterical contracture, and fibrous contractures rapidly develop, though this hardly ever occurs in hysteria. Marked atrophy is often present in the paralysed limbs in contrast with its complete absence or the very slight amount caused by disuse in hysterical paralysis. The vasomotor and trophic changes and excessive sweat are never seen in hysteria.

Organic disease may be simulated by the exaggerated tendon reflexes, the atrophy and occasional hypotonus, the alteration in electrical and mechanical excitability, and the presence of vasomotor and trophic symptoms. But the changes are generally easily distinguished from those occurring after an injury to a peripheral nerve, as the paralysis and anæsthesia do not follow the distribution of any nerve, muscular atrophy is never so extreme, the tendon reflexes are exaggerated instead of lost, and instead of the reaction to degeneration, the muscles may show exaggerated excitability to electrical stimulation. An upper neurone monoplegia generally affects a whole limb instead of a small segment; initial flaccid paralysis is followed by spastic paralysis, instead of flaccidity and spasticity often developing simultaneously in different muscles; the posture and gait are characteristic, and an extensor plantar reflex is present when the leg is affected.

Localised tetanus results in extreme hardness of the affected muscles, which are subject to very painful spasms in addition to the constant contraction; attempts to overcome it cause great pain, and it persists to an even greater extent under anæsthesia. I am,

however, inclined to think that some cases diagnosed as reflex neuroses by too enthusiastic disciples of Babinski and Froment have been examples of localised tetanus.

The etiology of Volkmann's contractures is quite distinct: the affected muscles are of a much more wooden hardness, and their electrical excitability is diminished or lost, instead of being normal or exaggerated. Vascular obliteration may give rise to similar symptoms, but the pulse is lost, intermittent claudication and gangrene may occur, and the application of heat has no effect on the pulse, whereas in reflex conditions the diminution in amplitude of the pulse on the affected side disappears.

It is clear, therefore, that these conditions are neither hysterical nor due to organic disease of the nervous system. They are not vascular in origin, as might be assumed from the cold, discoloured, and often swollen limb, and the diminution in the maximum oscillation of the pulse when compared with the opposite side, as the blood pressure is unaltered, and the vasomotor changes often extend a considerable distance above the injury.

It has also been suggested that the muscular atrophy is simply a result of disuse. But it occurs much too rapidly, and there may be no disuse of the muscles at all. Thus great atrophy occurs in the muscles of the forearm in cases of rheumatoid arthritis, in which the patient has never ceased to use the limb. On the other hand, little or no atrophy need occur in complete hysterical paralysis after months or years. The muscular atrophy around a tuberculous joint varies with the condition of the joint, and not with the amount of disuse, as the muscles may actually grow when the disease has been cured, although they are still at complete rest; and extreme atrophy occurs if a wrong diagnosis has been made and the patient is made to take exercise.

The possibility of an ascending neuritis is ruled out by the anatomical distribution of the symptoms, the exaggeration of the deep reflexes, and absence of reaction of degeneration; moreover, no anatomical evidence has been offered in support of this view.

Vulpian, in 1886, was the first to suggest that reflex atrophy and spasm could occur; his views were accepted and his observations confirmed by Charcot. All the symptoms can be explained by this theory. The increased tendon reflexes are due to a direct reflex; the vasomotor symptoms are due to a reflex spasm of the vessels and not to vasomotor paralysis, as the operation of stripping a muscle of its sympathetic fibres, which has been performed for the pain of causalgia, produces vaso-dilatation, a rise of temperature in the limb, and a rise of blood pressure of 20 to 40 mm. of mercury, whereas in the reflex neuroses there is vaso-constriction, a fall of temperature, and no change in blood

pressure. A reflex explains the spread of vasomotor and other symptoms far above the injury and even to the opposite limb; thus, in addition to the local effect caused by an injury of the hand, symptoms involving the muscles of the shoulder, and a slighter degree of vascular changes and excessive sweating of the opposite hand may occur.

The exaggerated muscular irritability to mechanical excitability, and the slow muscular contraction which occurs, are not due to direct reflexes, but are secondary to the vasomotor changes, as they are most marked in cold weather, when the limb is coldest, and disappear if it is warmed and the blood supply is increased. When a leg is affected, the plantar reflex may be lost, but this also reappears on warming the foot. The muscular atrophy may be due to the effect of afferent impulses from the injured or diseased parts on the nerve centres in the spinal cord, or possibly it is also in part secondary to the vasomotor changes.

Suggestion is quite useless in the treatment of reflex neuroses. They improve very slowly, but continuous extension and methodical and gentle mobilisation, together with whirlpool baths, heat, and especially diathermy, hasten recovery. Forcible manipulation of contractures under anæsthesia and tenotomies only do harm, but the excision of cicatrices with liberation from the subjacent tissue is sometimes useful.

A. F. HURST.

NERVE WOUNDS. Symptomatology of peripheral nerve lesions (299) **caused by war wounds.** J. TINEL. Preface by Prof. J. Dejerine. Authorised translation by FRED. ROTHWELL, B.A. Revised and edited by Cecil A. Joll, M.B., M.S. Pp. xii+317, with 323 figs. Baillière, Tindall & Cox, London. 1917. Pr. 15s. net.

WE have already expressed our high opinion of the value of this book in our review of the French edition when it first appeared (*v. Review*, 1916, xiv., p. 479) and have no hesitation in stating that good service has been rendered to neurology in its translation into English, as it places in the hands of those who do not read French one of the most important books upon lesions to the peripheral nerves which have yet been published. It is admirably adapted to the needs of the daily work of our military hospitals. It is based on a remarkably wide knowledge of nerve injuries, and presents a wealth of symptomatology usually clearly expounded and scrupulously exact. The illustrations are numerous, and have been reproduced with great care. The importance of a detailed knowledge of the anatomy, physiology, and histology of nerve is constantly emphasised in order that the facts may be correctly interpreted; and it is pointed out that a good clinical neurological knowledge is impossible without exact anatomy.

It must be a cause for satisfaction to all interested in neurology to know that the French edition was published before the lamented death of the late Prof. Dejerine, as it represents a résumé of the investigations conducted by his fellow-workers of the Charcot Clinic. A book such as this, bearing the name of Prof. Dejerine on its title-page, requires no further recommendation.

SHELL SHOCK. G. ELLIOT SMITH, M.D., F.R.S., and T. H. PEAR, B.Sc. (300) Pp. xi+133. 1917. Manchester, at the University Press, and Longmans, Green, & Co., London and New York. Pr. 2s. 6d. net.

A SHORT, concise, and essentially readable account of what is popularly termed "shell shock" is to be found here. It is pointed out that a more appropriate term would be "war strain." It is a condition characterised by instability and exaggeration of emotion rather than by ineffective or impaired reason, and involves no new symptoms or disorders. The most obvious phenomena are undoubtedly the disturbances of sensation and movement, and may vanish as suddenly and dramatically as they appeared. On the other hand, many tend to get worse, and if left without attention are apt to become stereotyped into definite delusions and hallucinations. The author urges strongly the necessity of treating such cases in special hospitals where their symptoms do not call for special remark, and are properly understood. The folly of isolating such cases is emphasised. Hypnotic treatment, when used with skill, discretion, and discrimination, has its place in the treatment of shell shock and similar conditions both in the acute and chronic stages, but in long-standing cases, especially with ante-war worry or emotion, hypnosis alone is of relatively little use, and may be positively harmful.

"The thousands of cases of shell shock which have been seen in our hospitals have proved, beyond any possibility of doubt, that the early treatment of mental disorder is successful from the humanitarian, medical, and financial standpoints. At present the State makes every provision for the insane after they have become insane, but no effort is made to prevent their becoming so. All that the asylum officials can say to such cases is—'Go away and get very much worse, and then we shall be allowed to look after you!' What is required is a psychiatric clinic 'staffed by skilled specialists who are familiar with the diagnosis and treatment of early and incipient mental disorders, not only with that of advanced insanity.' The chief functions of such a clinic would be:—

"1. Attendance on the mentally sick.

"2. Provision of opportunities for personal intercourse between patients and psychiatrists in training.

"3. Theoretical and practical instruction of students.

"4. Advising general practitioners and others who are faced with difficult problems arising in their daily work.

"5. Serving as a connecting link between investigation in the large asylums, and that in the anatomical, pathological, bacteriological, biochemical, psychological, and other laboratories of the universities.

"6. Scientific investigation of mental and bodily factors concerned in mental disease.

"7. Furtherance of international exchange of scientific knowledge concerning mental disorder by the welcome accorded to visitors from other countries.

"8. Dissemination of medical views on certain important social questions, and the correction of existing prejudices concerning insanity.

"9. When necessary, the after-care of the discharged patient."

THE CONDUCTION OF THE NERVOUS IMPULSE. KEITH LUCAS, (301) Sc.D., F.R.S. Revised by E. D. Adrian, M.B., M.R.C.P. Pp. xi+102, with diagrams. Longmans, Green, & Co., London. 1917. Pr. 5s. net.

THIS is the third of the series of monographs on physiology edited by Prof. E. H. Starling, the aim of each monograph being to give a succinct account of our present knowledge of different branches of physiology by those who have contributed in greater or less degree to the attainment of our present position.

This, the third monograph of the series, is based on a series of seven lectures delivered by Keith Lucas at University College, London, in the spring of 1914. These lectures were founded as a memorial to Page May, and their subject was the phenomena of conduction in nerve. They are published here in thirteen chapters, of which eleven had been completed by Keith Lucas himself by July 1914. At the outbreak of war he offered his services to his country, and was posted to the Royal Aircraft Factory at Farnborough. He was solely occupied with problems of flying from that time until the 5th October 1916, when he was killed in an aeroplane accident. The two remaining chapters have been written by Dr Adrian, one from notes left by Lucas, and the other, upon central inhibition, from a few notes and a list of references which show the main lines of the argument he intended to develop, and which Dr Adrian believes "does not misrepresent his views on the subject, though he may not have intended to publish these views until the experimental evidence was more complete."

The physiology of nerve conduction is most complex and difficult to investigate. The problem here to be determined is how far the phenomena of conduction in a peripheral nerve may

be made the basis of the understanding of conduction in the central nervous system. The experimental evidence obtained by Lucas is here described in detail, pure speculation being largely avoided, and the central nervous system is pictured as a network of conductors having different refractory periods, communicating through regions of decrement, easily fatigued, and capable of setting up a train of impulses in answer to a single stimulus.

Obituary

PROFESSOR J. DEJERINE.

INTERNATIONAL science has suffered a grievous loss in the death of Professor J. Dejerine, of Paris, on 26th February 1917. That wonderful galaxy of neurological "stars" in the Parisian school—Brissaud, Raymond, Dejerine, Babinski, Pierre Marie—who succeeded Charcot, has been sadly depleted by the inevitable advance of the years, and with the passing of Dejerine a vacancy is made which can scarcely be filled.

Dejerine was born in 1849, and graduated in 1879, with a thesis on the pathology of ascending paralysis. From the outset of his professional career he devoted himself to the study of neurology, and carried to the highest pitch of perfection the clinico-anatomical method of research, which has achieved most valuable results. In other words, he was a clinician as well as a pathologist, hence all his work was characterised by that practical importance which is not always manifest in the labours of the devotee to pure science. Undoubtedly his name will be permanently associated with the "*Anatomie des Centres Nerveux*," a remarkable production, whether we consider the organisation of an enormous mass of material, or the lucidity with which the conclusions are drawn, or the striking aid to comprehension furnished by a large number of fine drawings and diagrams. Similarly, his "*Sémiologie du Système Nerveux*," particularly its second edition, touches the high-water mark of lucid clinical representation of the facts of neurology. These two works alone are sufficient to establish the reputation of any worker in the field of neurology, but they scarcely indicate more than a tithe of Dejerine's output. Apart from many other books and brochures, the transactions of the Société de Neurologie

de Paris for the last twenty years, in fact since its commencement, provide proof of the fertility of his genius which astonishes the reader.

No one who has had the pleasure of seeing Dejerine in his historic Charcot clinique at the Salpêtrière, who has gone round his wards with him there, is likely to forget the experience—the charm of his manner, the ease with which he called on his store of clinical experience, the rapidity and certainty of his diagnosis, the persuasive and encouraging element in his treatment of functional cases, the friendly bearing towards every variety of patient.

Our colleagues of the French school know with us that neurology the world over is the poorer by the loss of Professor Dejerine, for though he had little English, his supereminence brought workers from every land to attach themselves for a time to his service, and they in their turn carried away with them an inspiration for research, the abiding value of which cannot be estimated in words. It is no mere figure of speech to feel and to know that the influence of such men as Dejerine was lives after them.

S. A. K. WILSON.

LIEUT.-COL. FROUDE FLASHMAN.

THE late Lieutenant-Colonel Froude Flashman was a graduate of the Sydney University, having attained his Bachelor's Degree in Arts and Science, and that of Doctor in Medicine. After graduation in Medicine he was for one year Resident Medical Officer at the Royal Prince Alfred Hospital, Sydney, and in 1895 received appointment as Junior Assistant Medical Officer in the Lunacy Service in New South Wales. He early evinced interest in pathological work, and when it was decided to establish a central pathological laboratory for research work in conjunction with the separate clinical laboratories already existing at the various mental hospitals, he was appointed to take charge. As a result of his advocacy this laboratory was located in the medical school of the Sydney University, Dr Flashman being of opinion that material benefit would accrue by association with the scientific staff there. His work was first devoted to neurological researches, and in collaboration with Professor Wilson he made many preparations of the brain. In 1903 he obtained leave of

absence to enable him to visit the chief centres of study in Europe, and he became acquainted first hand with their methods and the most advanced thought in matters pertaining to neurology and pathology of mental diseases. Amongst other places he visited the Pasteur Institute in Paris, Koch Institute in Berlin, and the Lister Institute in London. On his return he wrote a report embodying his impressions, and made valuable suggestions as to the future policy and methods that should be adopted at the Lunacy Department Laboratory, New South Wales. As Director of the Laboratory he was able to put these into operation, and within the next few years his reports published under the name of the laboratory attracted world-wide notice, particularly in regard to his investigation of the brain and skull of the Australian aboriginal. His work included the examination of the parietal regions of the brain of the aboriginal, the area striata of the occipital lobe, the general morphology of the brain and the relation of brain to skull, a very full account of the external and internal features of a microcephalic idiot, and he was the first to carry out the Wassermann reactions in Australia. He had also commenced a series of studies of the medullated tracts and nuclei of the brain stem of marsupials.

In 1910 he went into private practice as a consultant, but still retained his connection with the laboratory in the position of Director. He continued his researches, and edited reports of the laboratory published from 1902 to 1916, including not only the results of his own work, but that carried out in the various mental hospitals.

He acted at various times as lecturer at the Sydney University in Pathology, Neurology, Bio-Chemistry, and Mental Diseases.

Soon after the wounded Australian soldiers began to arrive in England, Dr Flashman was appointed O.C. of the convalescent depots there. With remarkable energy and foresight he got together a competent staff and an organisation with a central office at Horseferry Road to deal with the thousands of his countrymen, which he rightly anticipated would eventually have to be dealt with in England. Eventually he was appointed physician to Wandsworth Hospital, and afterwards went over to Boulogne, where, after some weeks' service, he caught a severe chill, which developed into pneumonia, from which he died after a brief illness.

Review

of

Neurology and Psychiatry

Original Articles

THE HISTOLOGY OF DISSEMINATED SCLEROSIS.

By JAMES W. DAWSON, M.D.

WITH A

PRELIMINARY COMMUNICATION

By the late ALEXANDER BRUCE, M.D., LL.D., and JAMES W.
DAWSON, M.D.

V.

PATHOGENESIS AND ETIOLOGY.

INTRODUCTION.

(1) THE NATURE OF THE PATHOLOGICAL PROCESS.

1. Developmental.
2. Inflammatory.

(2) ITS ORIGIN.

1. In the Neuroglia.
2. In the Nerve Elements.
3. In the Blood - vessels and Lymphatics.

(3) ETIOLOGICAL FACTORS.

1. Developmental.
2. External.
 - (a) Trauma.
 - (b) Psychological Shock.

(c) Chills.

(d) Exogenous Intoxications.

(e) Infections and Endogenous Intoxications.

(4) MODE OF ACTION OF THE CAUSAL AGENT.

1. Irregular Distribution and Circumscription.
2. Further Advance of the Process.
3. Modifying Factors.
4. Route of Conveyance to the Nervous Tissues.

INTRODUCTION.

To record these observations is the chief object of this paper: the secondary, but more difficult, task is to try to interpret them and to correlate the various conceptions which emerge in the process. It may be stated at the outset that no satisfactory explanation of the pathological process in all its bearings has yet been put forward, and that all that can be done here is roughly to estimate the factors which have been at work. It has been already pointed out that different investigators have given a different meaning to the same histological picture, while others, working solely or largely with individual elective staining methods, have laid stress on the feature of the process which that staining method rendered prominent. One important group, working with glia methods, considers that all the changes are subordinate to the primary glia proliferation. Another group, working with medullated sheath methods, equally forcibly maintains the primary parenchymatous origin of the process, and still others, working with diffuse stains, see in the changes in the blood-vessels the key to the whole process. From the histological point of view, therefore, the most important problems centre round the question of the rôle which falls to the various tissue elements in the origin of the process. From a clinical point of view, on the other hand, the question of the nature of the process is of more interest.

In the introduction it was briefly noted that it was necessary to discriminate between the nature of the process underlying disseminated sclerosis and its origin. In its nature it is due either to developmental causes, *i.e.*, those inherent in the individual, or to inflammatory causes, *i.e.*, those due to infections, intoxications, circulatory disturbances, &c., — these two groups not being necessarily exclusive, as will be pointed out later. In its origin the primary change may arise in any of the constituent elements of the nervous tissue—the neuroglia, the true nervous elements, or the blood-vessels. The views as to the developmental nature of the process are usually related to defects in the glia tissue, though a few writers refer to a “congenital degenerescence” of the true nervous elements. The views as to the inflammatory nature of the process are related again to primary changes in any of the tissue elements, and the factor or factors which bring the

inflammatory changes into operation are admitted to circulate in the blood-vessels or lymph channels, and to exert their action primarily on the glia, the myelin sheath of the nerve fibres, or on the blood-vessel wall itself.

The two views as to its nature strike at the very root of the chief difficulties met with in explaining the evolution of the morbid changes. The inflammatory nature of the process, in some form, was admitted by most of the earlier observers, who traced it chiefly to a primary change in the glia. This theory was later upheld, especially by French writers, who, however, traced the process chiefly to inflammatory changes in the vessel walls, but such an explanation has appeared less certain since the works of Strümpell and Müller. These writers stated that the special evolution of the disease, with its frequent marked remissions followed by aggravations without apparent cause, was difficult to reconcile with the view of a toxic or infectious cause alone having engendered the condition. They also point out that inflammation as a rule alters rapidly the axis cylinders and ganglion cells, which in disseminated sclerosis are frequently preserved, and, further, that inflammatory phenomena are often very difficult to trace. Recent research has greatly diminished the importance of these arguments by showing both that the long duration of the affection takes from the negative finding of inflammatory phenomena much of its significance, and that, even in disseminated sclerosis, the ganglion cells and axis cylinders frequently perish.

For more than a quarter of a century after Charcot gave the first clinical and anatomical picture of the disease, it was looked upon as a distinct morbid entity, a chronic disease in which certain characteristic clinical and anatomical features were always present. Many later writers, however, have related it to a disseminated form of myelitis. They assert that previously only the final stages of the disease have been considered, and that by suitable staining methods, areas may frequently be observed in the same case in all stages of development, from an area of inflammation into an area of sclerosis, from which all traces of inflammation have disappeared.

To this argument it is replied that there exist two forms of the disease: a primary chronic form due to a malformation of the glia, and a secondary myelitic form *en rapport* with infection and intoxication. It is admitted, however, that in primary disseminated

sclerosis the development of the focus may begin under the reaction of external factors as *agents provocateurs*, but the disease is in no sense dependent upon these. In opposition to this, it is asserted that the separation into primary and secondary disseminated sclerosis is an artificial and arbitrary one, and that no clinical or anatomical distinction exists between the two. An intermediate position is taken up by those writers who reconcile the inflammatory theory with the developmental one. They admit that congenital anomalies of the glia may become the point of origin of primary proliferations of the glia, but they believe that the blood-vessels distribute some toxi-infectious agent which settles there where the glia is abnormal, and thus calls this glia proliferation into being.

Turning now to the histological observations, it is necessary to ask: Do these throw any light:

(1) *Upon the Nature of the Process?*—Do the areas arise solely upon the basis of a gradually increasing glia hyperplasia, or on the basis of an inflammatory reaction, or on both? Further, are there sufficient grounds for distinguishing between the two?

(2) *Upon the Origin of the Process?*—If we admit a primary form of disseminated sclerosis of developmental nature, we have answered the question of its origin in the glia; but if we say that the underlying process is of an inflammatory nature, we thereby also say that the blood-vessels or lymph channels carry the ultimate causal factor to the tissues, but must further decide on which tissue element there is the first evidence of its action.

(3) *Upon the Etiological Factors Postulated*—chill, trauma, psychic shock, intoxications, infectious diseases, &c.?

(4) *Upon the Mode of Action of the Causal Agent*, and the other questions which this consideration involves?

For the purposes of our argument it is assumed that the cases investigated were cases of disseminated sclerosis. This follows (1) from the clinical notes submitted; (2) from the macroscopic findings of disseminated areas in spinal cord and brain, with the characters usually ascribed to this disease; (3) from the recognised microscopic characters of these isolated or confluent areas in which were found absence of the myelin sheath of the nerve fibres, and varying degrees of glia proliferation, blood-vessel changes, and persistence of axis cylinders.

(1) NATURE OF THE PATHOLOGICAL PROCESS.

1. *Developmental.*

Müller has strongly upheld the view of the developmental nature of disseminated sclerosis, and has emphasised the distinction between the primary and secondary forms of the disease. Weigert's work showed that both from the morphological and biological points of view the neuroglia reacts as a true connective tissue. Müller's wider conception of this idea leads him to see that the ultimate product of processes entirely different in their pathogenesis, but presumably all giving rise to focal disease of the parenchyma, may show disseminated sclerotic patches. He divides them, on the one hand, into those processes which always occur as the direct result of exogenous factors and lead to multiple focal degenerations and inflammations, and, on the other hand, into a morbid condition which has one uniform circumscribed pathogenesis, being due not to any known external cause but to a congenital abnormal disposition of the glia. Among the former, he states, are those "comparatively rare" processes which have resulted in injury to the nervous tissue due to some apparently primary blood-vessel condition, *e.g.*, syphilitic or arterio-sclerotic disease of the vessels, or to a toxi-infective inflammation in the form of a disseminated myelitis or encephalitis. In all these cases the histogenetic terminal product is a secondary disseminated sclerosis in the sense of Schmaus and Ziegler. In contrast to these is the "comparatively common" disease caused by congenital disturbances of development, and termed by Schmaus and Ziegler primary disseminated sclerosis. This form, which presents distinct clinical and anatomical signs and may be looked upon as a multiple gliosis (Strümpell), is the only true disseminated sclerosis.

The essential anatomical signs of this condition are the following: (1) the foci are situated only in those parts of the nervous system which normally contain much glia; they tend to develop symmetrically, and are often of considerable size, *e.g.*, in the cord they may occupy the whole transverse section; their relation to the blood-vessel can be explained by the layer of glia—the glia limitans perivascularis—normally around the vessels. (2) Microscopically the foci consist of an excessive proliferation of glia, leading to a dense tissue which is never areolar: there is comparative integrity of the ganglion cells and axis cylinders in

the area and a marked degeneration of the myelin sheath, with no secondary degeneration; and there is never evidence of a primary disease of the blood-vessels within the area. The foci, stained by means of Weigert's myelin sheath stain, appear clearly defined and as if punched out, but, stained by Weigert's glia method, the areas have not the same abrupt transition to the normal tissue—the proliferated glia becoming gradually lost in the periphery. In this circumstance Müller found an argument for the primary glia origin of the process. He bases much of his description of the areas on sections, stained by Weigert's glia method, lent to him by Weigert himself. Sections so stained bring out very forcibly the enormous proliferation of the glia, and give the impression of a primary glia growth so out of proportion to the relative integrity of the ganglion cells and axis cylinders as to contradict any mere substitution proliferation. Müller thinks it very striking that this colossal glia proliferation—the maximum found in pathological conditions (Weigert)—should be met with in a disease in which there is relative integrity of the true nervous tissue.

The clinical signs are also typical, and Müller thinks that secondary disseminated sclerosis can never successfully simulate Charcot's classical picture in the marked distinctness of each symptom and the peculiarly characteristic course. In spite of the great variableness in the symptoms, which may simulate widely-differing diseases of the brain or cord, the diagnosis can always be established by a study of certain individual symptoms and groups of symptoms, and of the course of the disease. This study will reveal the fact—at first sight paradoxical—that the fundamental features of the clinical condition are in the great majority of cases absolutely the same. There is usually no real exciting cause: it may commence suddenly, but more commonly has a subacute or chronic onset; and it shows sudden exacerbations with marked remissions, but is, on the whole, a progressive and chronic disease.

Müller holds that the relationship between infectious diseases and disseminated sclerosis has apparent justification only in cases where the one disease immediately precedes the other, or where there is a direct transition of the one into the other. True disseminated sclerosis shows a marked preference for youth, and the fully-developed syndrome is very rare in childhood during the years when infectious diseases are most common. Cases showing a close time-relationship are very numerous, and clinical experience

shows that external factors are quite insignificant in the etiology of disseminated sclerosis. Further, the idea of a direct causal relationship appears incompatible with a satisfactory explanation of many of the characteristic features of the course of the disease. Disseminated sclerosis runs a chronic course, and Müller asks if the typical exacerbations in its course are to be explained by inferring that the various exciting agents of scarlet fever, diphtheria, &c., circulate for months or years, and become deposited in the foci to give rise later to fresh infection. On the same grounds Müller thinks it unlikely that disseminated sclerosis is caused by the action of metabolic products derived from bacteria (toxins), as in post-diphtheritic nephritis or polyneuritis. For this we would have to suppose that the products of metabolism from absolutely different bacteria and of entirely different chemical composition could give rise to absolutely similar foci, and also that a paroxysmal increase and decrease in the action of the toxin accounts for the exacerbations and remissions. He further thinks that such meta-infectious diseases are more likely to produce diffuse and system diseases than focal processes. True disseminated sclerosis, just as Friedreich's ataxia and psychoses due to congenital disposition, may develop at the conclusion of an acute infective disease, but all recognisable exogenous factors, which in a small minority of cases have a definite time-relationship with the commencement of the clinical symptoms, are capable only of acting as *agents provocateurs*—given an existing predisposition to the disease—making manifest or aggravating the condition. Infectious diseases may, however, give rise to a disseminated disease of the central nervous system, which may develop into a secondary disseminated sclerosis, but its pathogenesis, course, and histological details differ from true disseminated sclerosis. So-called acute disseminated sclerosis is to Müller simply a disseminated affection of the central nervous system, which really belongs to disseminated myelo-encephalitis. In the rare cases in which it is difficult to make a pathological diagnosis between true disseminated sclerosis and the secondary forms, a consideration of the whole clinical condition and especially of the course of the disease will decide.

Müller points to the fact that, so far as we are aware, there are no exogenous factors which seem to produce in any other organ the development of a process comparable in any way to disseminated sclerosis as a strong argument in favour of its

endogenous or developmental origin. By tracing true disseminated sclerosis to "abnormal congenital conditions," he simply differentiates it from forms due to recognised exogenous factors. He is in no way satisfied with this explanation, and states that further investigation must seek to discover the organic basis of such abnormal congenital conditions.

It is thus seen that the explanation of a "multiple gliosis" in Strümpell's and Müller's sense is very similar to that given for certain groups of tumours, which must be related to embryonal defects. Ritchie, discussing the etiology of tumours, states that the soundest ground for assigning congenital defect exists when the tumour arises in some part of the body where at some stage of foetal life the cells of one tissue must push aside those derived from another in order to attain their ultimate natural position: the sequence being that during the building up of the body the cells thus detached or pushed aside, after being embedded for a longer or shorter period, take on a vegetative activity and assume the characters of a tumour. Müller claims that the favourite sites for the development of this gliosis are those which in the embryonal period show specially active processes of growth, or where two surfaces come together and fuse in later development ("Kielstreifen"), or where the marginal zone of the embryonic nervous system is pushed inwards by the ingrowing vessels. At such sites developmental disorders, such as germ invagination or detachment, might set in very readily. The ventricular surfaces and the peri-central tissue normally contain much glia, the postero-median septum and the optic chiasma are formed by such "Kielstreifen," and the marginal glia zone and the peri-vascular glia zone represent tissue carried inwards during development. Areas are, therefore, not necessarily related to blood-vessels, and when so related it is in virtue of the peri-vascular glia layer which surrounds their adventitial sheath. The frequent symmetry of the areas is related similarly to the glia and not to the distribution of a toxin by the blood-vessels.

The sequence of the process, as has already been pointed out, is a gradually increasing hyperplasia of the abnormal glia in these sites of predilection. This results in a disappearance of the myelin sheath of the nerve fibres, partly through direct compression, and perhaps indirectly by derangement of the circulation. No fat granule cells need necessarily appear at first, but in the zone

surrounding the compact area there may be a secondary reaction to the products of degeneration, leading to a combination of primary and secondary proliferation with peripheral progress of the process. Here fat granule cells would appear as indications of a more recent process but by no means of an exogenous inflammatory one. The blood-vessels within the sclerosed area also show changes which are entirely secondary to the sclerotic process, which, in consequence of the peri-vascular glia layer, is specially marked immediately around the vessel.

Areas in secondary disseminated sclerosis, on the other hand, are stated to be of an "areolar" type: there is much greater involvement of the axis cylinders and ganglion cells: there is seldom any sign of real sclerosis—only the remaining glia network is present or a slight consolidation of it, which sets in concentrically from the normal tissue, not excentrically from the abnormal glia predisposed to proliferation: the blood-vessel changes are marked, and the dependence of the areas on the altered vessels is striking; and the foci are, as a rule, smaller in size, are frequently followed by secondary degeneration, and are often limited to the cord. Clinically, also, the disease occurs in definite relation to varied toxi-infective processes: after a shorter or longer period the symptoms either steadily progress or gradually recede to more or less complete recovery; and there is an absence either of any striking fluctuations during its course or of any further relapses.

In all the six cases examined by him, Müller found, histologically, a complete absence of the undoubted components of inflammation, and, clinically, in eighty cases, an absence of toxi-infective processes in the anamnesis. He therefore felt justified in excluding exogenous factors and falling back upon developmental causes. It is difficult to criticise so important and careful a work as Müller has given in his monograph—the chapter on the diagnosis and differential diagnosis alone extends to a hundred pages. It is there stated that more than twenty different diseases of the nervous system may be simulated by disseminated sclerosis, that a typical case is one of the most readily recognised diseases of the nervous system, but that atypical forms constitute by far the greatest proportion. We are here more concerned with the histological data, and these, as given in the monograph, are very slight. Several staining methods are briefly mentioned, but reliance has been placed chiefly on specimens lent to him and

stained by Weigert's glia method, and also on Weigert's myelin sheath and Marchi-stained sections. The latter showed at the periphery of the areas an extending process, but all the areas examined in each case showed a dense, compact glia structure, and nowhere was there any indication of soft, *i.e.*, early areas.

In all the nine cases examined by me there have been present side by side with dense areas, or forming an outer zone to such, very numerous foci in which transitions could be traced from "early" areas with numerous fat granule cells—the criterion taken by many writers for the existence of an inflammatory reaction, and which for the present I accept—to areas of almost complete sclerosis in which there was no indication even at the periphery of an advancing process. But such complete sclerosis was comparatively rare: even in the most advanced case, many of the areas showed the presence of fat granule cells in the walls of the vessels both within the area and leading from it: and the great majority of the dense areas showed a central sclerosis, gradually becoming less as it passed into the normal tissue, and this transition zone gave the impression that had time been given it would have undergone an excentric spread comparable to the central gliosis. The end result, then, of all such areas would probably have been a compact gliosis in which were very fine meshes not in the least similar to those of the areolar areas described by Müller as characteristic of secondary disseminated sclerosis. The examination of many hundreds of areas has satisfied me that "early" areas—so-called areas of fat granule cell myelitis—can develop into areas of compact gliosis with all the characteristics of those described by Müller as typical of true, primary disseminated sclerosis. None of the cases could be described as "acute multiple sclerosis," taking this term in the sense Marburg and Dinkler have used it. In Case I., which has the shortest clinical course in the series, the illness had lasted fifteen months and death had resulted from diarrhoea and exhaustion. In this case no fewer than fifty different areas were examined by the Marchi method alone, and four-fifths of these showed fat granule cells distributed throughout the whole affected tissue. In the other areas these cells were limited to the periphery and to the vessel walls, while the centre of the area was composed of a dense glia feltwork with numerous axis cylinders in the fine meshes. In only two areas was there a complete absence of any such signs of inflammatory reaction.

In the very numerous areas examined by other staining methods, a similar structure could be demonstrated and in similar proportions, for the areas chosen for the Marchi method were taken irrespective of their macroscopic soft or hard consistence.

I am in agreement with Müller's statement that the glia tissue had frequently not the same abrupt definition as the demyelinated tissue. Though not using Weigert's glia stain, it was often quite evident that the myelin sheaths at the margins of the area, in longitudinal sections, were thrust asunder by proliferated glia tissue. This, however, need not necessarily be explained on the ground of a primary glia proliferation. I am further in agreement with Müller that the most frequent sites are the peri-ventricular and peri-central tissue, around the postero-median septum, in the lateral columns, and with the marginal glia zone as a base. Why certain parts are predisposed it is difficult to say, and this question must be discussed later. More easy to understand is the frequent remarkable symmetry emphasised by Müller. This has been entirely confirmed, especially in cases where there was an almost complete transection of the cord, when the symmetry was perceptible in the marginal portions of preserved fibres, and again when both the lateral columns were affected and marginal zones were left corresponding to the dorsal cerebellar tracts. Müller's explanation of this symmetry has been referred to, but it may also be explained by the possibility of the two halves of the cord being exposed at the same time to a diffusely acting agent in the blood-vessels.

Müller's essential arguments are: (1) that the participation of the blood-vessels within the area is only a secondary one, and (2) that the glia proliferation is far more than reparatory. In discussing the origin of the process we must refer to both of these points and also to Strümpell's view that the vessels throughout the body, not solely in the central nervous system, would be affected were it an inflammatory process. But it may be stated here that Strümpell and Müller's view of a "multiple gliosis" is scarcely tenable for the cortical areas, which have been proved not to consist of proliferated glia fibrils, and for areas in the peripheral nerves. The latter, however, if their existence can be demonstrated, might well be analogous formations—consisting of a proliferated, interstitial tissue whose origin is in the Schwann's sheath, in virtue of its ectodermal derivation from the neural crest.

From a comparison of the histological study with the above statements, it will be seen that I am not in agreement with Müller's view that the areas in disseminated sclerosis arise solely on the basis of an increasing glia hyperplasia, and that they can be always separated from those arising on the basis of an inflammatory reaction. In this study there is overwhelming evidence that the great majority of the areas have arisen on an inflammatory basis and that a small minority have arisen on the former basis. The end result of both is a tangle of glia fibres. During the process of the gradually increasing glia hyperplasia it is possible, especially in the lateral columns of the cord, to differentiate this mode of formation of a sclerosed area, and I relate such not to a developmental defect of the glia, as Müller and Strümpell have done, but to a special reaction in the glia, according to the nature and intensity of the causal factor. (see p. 409). We have therefore not two affections, or necessarily different stages of the same process, but one causal factor which probably acts with varying intensity.

2. *Inflammatory.*

Having therefore given adherence, on the grounds of an exhaustive study of nine cases of disseminated sclerosis, to the inflammatory nature of the process underlying this condition, it must now be considered briefly what is meant by inflammation in the central nervous system.

It is usual to classify the varying histological pictures in the processes termed "inflammatory" in the central nervous system into parenchymatous and interstitial, analogous to the processes in the glandular organs of the body. But the conception of a purely parenchymatous inflammation in the central nervous system has little anatomical support, for the evidence of changes of a regressive nature in the nerve cells, axis cylinders, and myelin sheaths is accompanied by evidence of progressive phenomena in the other tissue elements. It is not possible, therefore, to draw any clear distinction between simple tissue degeneration and inflammation, and it is usual to designate as "myelitis" both the processes, which are from the beginning distinguished by inflammatory exudations and cell infiltration and those which begin as degenerations and only in their further

course are connected with pathological exudation or proliferative processes.

When, therefore, the inflammatory nature of the process is referred to, it is as a reaction process and one not necessarily associated with exudation of fluid and cell infiltration of the vessel walls. All changes which reveal any kind of progressive phenomena in any of the tissue elements are therefore included in this view of inflammation. Later the relations of disseminated sclerosis to acute and chronic myelitis must be referred to, but the widely varying views regarding the true nature of "acute myelitis" may here be touched upon. Many clinicians have given this name to all diseases of the spinal cord, the symptoms of which cannot be traced to an isolated affection of individual portions of the cord or systems of the cord. Others, before applying the term, make the additional condition that an inflammatory process should really be present. The conditions under which it is justifiable to entitle a disease "myelitis" are not yet defined either clinically or anatomically, and it is the anatomical substratum of the disease, when known, that usually defines the idea of the disease. Bastian, arguing largely from the similarity of the morbid changes to those occurring in the brain, which are due to thrombosis, has long maintained that the great majority of cases are really "thrombotic softening of the cord" rather than an infiltrative myelitis. An ischæmic softened area in the brain, in which great numbers of phagocytic cells, associated later with proliferation of the supporting tissue, are present, is not usually looked upon as an encephalitis. It is the custom to identify this finding by the name of "softening," and yet it is much rather a reactive condition that has set in secondarily, and is a secondary inflammatory reaction—secondary to the presence of the degenerated products. A similar condition may occur in the spinal cord as a result of thrombosis of the spinal vessels, and the reactive phenomena are here again not due to the primary cause which produced the tissue necrosis. On the other hand, as a result of toxi-infective agents, it is possible to get (1) typical cell infiltrations of the vessels and surrounding tissue; (2) simple degenerations of the tissue, in which the so-called inflammatory vascular changes may be absent; or, further, (3) actual inflammatory softenings of the tissue. In the latter two forms we again get later reactive changes in the

tissue elements, and these may be secondary to the tissue degeneration, or simultaneously called forth by the primary causal stimulus. Are such conditions to be termed inflammatory only when the primary blood-vessel wall changes are inflammatory? or is it not possible to recognise the complicated inter-reaction as an inflammatory process? Clinically, we cannot yet distinguish parenchymatous and infiltrative myelitis, nor these from acute inflammatory softening and acute thrombotic softening. Pathologically, it is sufficient to distinguish two main types in which myelitis may show itself: (1) Infiltration—a form to which some would limit the term “acute” myelitis; (2) softening—to which the term “myelomalacia” has been recently applied—a form which may be very varied in degree from the degeneration of a limited number of nerve fibres, in which the process is more probably subacute, to the necrosis of a large area, in which the process is more likely to be acute in onset.

(2) ITS ORIGIN.

It is necessary now to discuss the question of the structural element of the nervous tissue in which this inflammatory process has its origin. In the fully developed sclerotic area changes are evident in relation to the myelin sheath of the nerve fibre, the glia, and the blood-vessels. It is important to recognise on which tissue element the causal agent, circulating presumably in the blood-vessels or lymph sheath of the blood-vessels, first produces its effect.

1. *Changes in the Neuroglia Tissue.*

Charcot defined the histological picture of disseminated sclerosis as a chronic interstitial inflammation leading to a gradually increasing glia hyperplasia. This view is so frequently confused with that formulated by Strümpell, Ziegler, and Müller, in which there is also a gradually increasing glia hyperplasia, that at the risk of a too frequent repetition the distinction between the two must be emphasised. Strümpell's view is that abnormally placed glia cells take on a latent vegetative activity, and produce a multiple gliosis, while Charcot holds that an exogenous causal factor stimulates the glia anywhere within its range to a marked glia fibril formation. The sequence of the process appeared to him to be the following: the multiplication of the glia nuclei

and concomitant hyperplasia of the reticulated fibrils of the glia constitute the initial fundamental fact and necessary antecedent; the degenerative atrophy of the nerve elements is consecutive and secondary, and the hyperplasia of the vessel walls plays merely an accessory part. Disseminated sclerosis is, therefore, looked upon as a primary and multilocular chronic interstitial myelitis and encephalitis. The contention of a primary glia change is based on the presence of glia hyperplasia, while as yet there is little or no evidence of either an alteration of nerve fibres or changes in the blood-vessels, and also on the fact that at the periphery of the areas, where presumably the morbid process is still active, there is a marked increase of glia cells; here, also, are the evidences of the strangling of the nerve fibres by the glia fibril formation. The substance of the argument of those who support this view seems to lie in the acknowledged fact that in many areas the glia proliferation is far in excess of that required as a mere reparatory or substitution process, and that it must therefore be looked upon as a productive primary stimulation. Weigert, whose views on such a subject necessarily carry great weight, believes that the maximum of pathological glia fibril formation is seen in this disease.

The presence of areas, especially in the lateral columns of the cord, of a gradually increasing glia hyperplasia, has already been indicated, and their comparative rarity in this study emphasised. I am, therefore, not in general agreement with this view, seeing it applies to so few of the areas, but in isomorphous sclerosis it is probable that the reaction phenomena may be all the more marked when the tissue is not greatly altered, and this would account for the colossal glia proliferation. In the changes in the optic nerve also, there were evidences of a primary change both in the connective-tissue elements of the endoneurium and in the glia septa. Similar changes are described by Fleming in retro-bulbar neuritis in cases of intracranial tumour, and are ascribed by him to a tonic condition of the cerebro-spinal fluid. In other areas, especially where there was a gradual atrophy of the myelin sheath, an indirect action of the sclerosis on the nerve fibres could be traced, possibly by the sclerosis limiting the expansion of the blood-vessels, and interfering with the nutrition of the nerve fibres, but it is far from this to the direct compression causing destruction.

In referring to the histological study it will be noted that I seem to be in further agreement with the supporters of this view in looking upon the first change as being evidenced in the glia tissue. There it has been stated that an enlargement of the protoplasm and protoplasmic processes of the normally existing spider cells was the first change visible, but this must be referred rather to the difficulty of recognising, by such staining methods, an early change in the nerve fibre, and a change in isolated glia cells must be looked upon as being in the great majority of cases simultaneous with or possibly later than that in the myelin sheath. It has also been repeatedly stated in the histological study that the enlargement and proliferation of the glia cells can be traced amongst the normal fibres at the margins of the demyelinated tissue. This change and the presence of the nucleated peripheral zone of glia cells seem to us to be not necessarily a proof of the primary and essential change being related to the glia elements. Its possible significance will be referred to later in relation to the varying factors which may influence the development of the process, for it is important to recognise that the areas do not always develop proportionately.

A brief allusion may be made to the various functions attributed to the neuroglia. It is no longer held that the glia cells and their processes conduct the nutritive substance from the vessels to the nerve cells and fibres, and it is also probable that the neuroglia must be looked upon as more than a supporting structure or as a tissue element which serves to isolate the ramifications of the neurones. Lugaro has put forward the interesting and suggestive view that the glia serves to transform the products of metabolism of the nervous tissues and to render them inoffensive—the peri-vascular glia thus acting as a filter. Nageotte and Babes have both ascribed to the glia cells a secretory action in virtue of their derivation from epithelial cells.

2. *Changes in the Nerve Elements—Nerve Fibres and Ganglion Cells.*

Redlich, Huber, Storch, and others have contended that the "noxa" acts directly and primarily upon the nerve fibre before there is any trace of glia proliferation or vessel alteration. This

change may be purely degenerative or an actual inflammatory degeneration. The interstitial changes in the glia may be, therefore, on the one hand, secondary to the parenchymatous degeneration, or on the other, also productive—passing beyond the bounds of a substitution process. The view that the toxic agent has an affinity for myelin has been supported by numerous recent writers: *e.g.*, Marburg speaks of the process as essentially a “lecitholysis,” and Mott thinks it might be explained by the slow, limited, and localised action of some “lipolytic” ferment which attacks the myelin covering of the nerve fibres.

In our histological study it has been pointed out that numerous small areas occur in which there is no appreciable change but a demyelination of the affected tissue. This is well brought out in Weigert myelin sheath sections, but in normally-evolving areas it is more apparent than real, for sections stained by Heidenhain's iron-hæmatoxylin almost invariably show that in such small areas the slightest demyelination is accompanied by a commencing glia cell proliferation, which continued parallel to, or even exceeded in proportion, the destruction of the myelin sheath. On the other hand, numerous areas are present in which there is a complete absence not only of the myelin sheath but of all signs, in the presence of fat granule cells and proliferating glia cells, either within the area or at its periphery, of an extending process, and in such areas the abundance of the glia does not justify the name of sclerosis. Here, again, it is probable that varying factors have been at work to alter what may be looked upon as the normal evolution of a sclerotic area. Arguing, however, from the presence of such areas, I am in entire agreement with the view that the most constant and uniform change is the absence of the myelin sheath: this usually commences as an early swelling, varicosity, and faint staining, which passes into a finely granular degeneration.

The glia proliferation, in the great majority of the areas, is, therefore, called forth by two factors: it is immediately occasioned by the stimulant action of the “noxa” which caused the degeneration of the nerve fibre, and it is secondarily brought about by the degenerated products of the parenchyma. The latter effect is explained in part by the irritant action of these products and in part by the well-known conception of Weigert (*Wegall von Wachstumshindernisse*) that the constituent tissues of an organ

are usually in a state of equilibrium, so correlated to one another that no cell can disappear without its place being taken by hyperplasia of the surrounding tissue. Thoma has suggestively applied this conception to explain the comparative absence of glia in the cortical areas. He points out the importance of considering two factors in the sclerosis: (1) the proved resisting power that the ganglion cells and axis cylinders show, and (2) the non-resistance of the myelin sheath, and he thinks that these two opposing factors account for the proportionate development of a sclerosis. In the medullary rays and at the base of the radiations in the cortex, where the myelinated fibres lie close together, their extensive degeneration brings about a marked sclerosis, but in the actual cortical layers two unfavourable factors—few myelin sheaths and a trifling glia content—meet with a resistant factor in the numerous ganglion cells and axis cylinders, so that there results a sclerosis so limited that numerous authors state that it does not exist in the cortex.

Cajal and Marinesco, in relation to the satellite cells of the grey matter, have also developed Weigert's conception. It is maintained that the nerve cells and the glia cells develop parallel to one another, and that in the normal state there is established a nutritive equilibrium between these two elements. This equilibrium probably is maintained by the secretion of certain substances—elaborated by the nerve cell—which hinders the excessive development of the glia cells. The nerve cells and satellite cells, therefore, constitute a kind of symbiosis, but Mott thinks that the proliferation of satellite cells seen in acute toxic conditions is due to a failure of assimilative metabolic processes in the nerve cells as a result of the poison. There is, therefore, more nutriment at the disposal of the satellite cells, and they are thereby stimulated to proliferation.

In the description of areas in the grey matter, it has been shown that the involved ganglion cells in the cortex were frequently surrounded by proliferating satellite cells of various forms with numerous fine black granules in their protoplasm and branching processes (osmic acid), and at other times almost replaced by nests of proliferated satellite cells. In the grey matter of the spinal cord, on the other hand, no such satellite cells were ever found, and the ganglion cells seem there to undergo simply a gradual atrophy.

3. *Blood-vessels and Lymphatics.*

The general agreement of the areas, especially of isolated cerebral areas, with the topographical distribution of the blood-vessels has long led to the belief that these were primarily affected in the diseased process. The changes in the vessels may be expressed, however, in very various ways: a chronic inflammation of the walls of the vessel may affect the nutrition of the area supplied by it: primary thrombosis: or thrombosis secondary to irritation of the intimal lining, by toxins circulating in the blood, might lead to similar malnutrition: some effect, toxic or mechanical, on the intimal lining may lead to minute capillary hæmorrhages: a primary acute vascular change with peri-vascular cell infiltration—a true inflammation in the generally accepted sense of the term—might extend to involve the adjoining tissue: or, finally, the vessel wall changes might lead to adhesion and closure of the lymphatic sheaths with a subsequent lymph stasis in the tissues. Each of these views has its adherents, and it is necessary briefly to indicate the sequence of the changes.

Rindfleisch thought that the chronic changes in the vessel walls (the cause of which appeared to him quite unknown) was followed by an atrophy of the nerve elements from malnutrition and a secondary glia proliferation. It is evident that the areas in which such chronic vessel changes were found must have been old sclerotic areas, in which the changes may be the effect of the sclerotic process and not its cause.

Ribbert thought that the exciting cause of the inflammation circulates in the blood: that owing to its presence a clot is formed at some part of a small blood-vessel; and that at this point an irritation of the vessel wall is set up with a peri-vascular inflammation, which extends to involve the surrounding tissue—causing degeneration of the nerve fibres and an active proliferation of the glia. The liability to thrombosis after the acute specific infections is well recognised, and French writers especially have suggested that multiple thrombi form owing to an altered condition of the blood or of the vessel wall. In the area of supply of such vessels there would appear ischæmic degeneration, followed by phagocytic cells, and a substitution glia proliferation. It is pointed out that such areas, unless examined shortly after the thrombus formation, would reveal no trace in the vessel of the cause of the focal degeneration. Amongst recent

writers Siemerling and Raecke have attached considerable importance to the presence of capillary hæmorrhages. In all the areas examined by them, and especially marked in the cortical areas, were minute hæmorrhages which were looked upon as the first evidence of the inflammatory process, and the cause of the initial fibre degeneration.

During the course of this investigation, a large number of small isolated areas, both in the brain and spinal cord, have been cut in serial section with the object of tracing the possible presence of thrombosis or of capillary hæmorrhages. In a few instances, especially in the lateral vessels of the cord and medulla, there have been found aggregations of white cells and the presence of fibrin, which have been taken as indications of intra-vital thrombosis, but nowhere has evidence been present of organisation of such thrombi nor of alterations in the vessel walls in relation to them, nor have these been always in relation to sclerotic areas. Again, in close relation to the engorgement of the blood-vessels, both within and without the areas, small hæmorrhages have been found. These, however, show no changes, and were looked upon as probably the result of the respiratory difficulties before death. The vessel walls also showed no changes which would explain the hæmorrhages, nor were there any signs of inflammation around them. Keeping in mind the difficulties in recognising small thromboses after actual sclerosis has set in, and also the admitted long time that extravasated blood may remain unchanged in the nervous tissues, it is difficult to account for the origin of sclerotic areas in such primary changes. The absence of any histological evidence of changes in the vessel walls associated with thrombosis or hæmorrhages is quite incompatible with a primary vascular lesion in this sense.

To true inflammatory changes in the walls of the blood-vessels a primary significance has been ascribed by a very large number of writers. "In the early stages inflammatory alterations, with small-celled infiltrations, can be recognised in the vessel walls." "The primary vascular inflammation determines a peri-vascular embryonic infiltration." "In recent areas the reactionary vascular phenomena are entirely out of proportion to the myelin degeneration." Such statements, asserting the primary inflammatory character of the lesion in the vessels, can be found extensively throughout the literature of this subject, together with statements

which show that the "granular cell myelitis" is also taken as a proof of the presence of an acute inflammation in the vessel walls. The sequence of the process is either that the primary affection of the vessel wall is transmitted direct, by a progressive diffusion, to the surrounding tissues, causing a solution of the myelin and a reactive interstitial inflammation, or the agent causing the inflammation is restricted to the vessels and causes in these first of all alterations, through which are produced changes in the nutrition of the surrounding tissue, which are no longer inflammatory but purely degenerative.

It would *a priori* be thought natural that an irritative substance circulating in the blood would, in its filtration and diffusion into the tissues, stimulate the capillary endothelium and cells of the adventitia to proliferate, and that its first effect would thus be on the vessel wall through which it passed. In this investigation special importance was attached to this point, and a reference to the histological study will show that it was impossible to trace, except in rare instances, a primary proliferation of the capillary endothelium, or a primary increase in the nuclear content of the adventitia. The first cell infiltration of the vessel wall was one of fat granule cells in the adventitial lymph spaces secondary to the resorptive processes: this called forth a secondary proliferation in the endothelial and other cellular elements of the adventitia, and at a later stage there was a cell infiltration of lymphocyte-like cells analogous to those found in all chronic processes. In the event, therefore, of a disease-producing agent, either bacterial or toxic, being carried with the blood or circulating in the lymph sheaths, it must be assumed that this poison leaves the blood channels in so slight an amount or in such weak concentration that a recognisable injury of the vessel wall does not result. The only part played by the blood-vessels would thus be the bringing of the "noxa" to the tissues. Bielschowsky, who looks upon the vessel changes as entirely secondary, has come to the conclusion that only a "noxa" that has penetrated by the vessel into the tissue but has left the vessel wall intact has occasioned the process. Taylor, who was unable to find any trace of vessel changes in the examination of eight cases of disseminated sclerosis, thinks that it is perfectly conceivable that the manifestation of the toxic agent may occur without evidence of local inflammation in the

vessel wall. Marburg and other writers, who have described cases of "acute multiple sclerosis," have given this designation instead of "acute myelitis," "in virtue of the absence of any marked signs of actual inflammation in the vessel walls." The explanation of the numerous findings of such inflammation by other writers must be found either in the presence of areas of an acuter type than were observed by me, or in the possibility that areas were described at a stage when the secondary infiltration of fat granule cells and the cellular reaction to their presence had occurred.

Alongside of the vessel lesions and dependent on them, Borst, Schmaus, and other writers have described disturbances of the lymph circulation. The sequence of the process is a little difficult to follow, for the change may be evidenced by a slight α edema and rarefaction of the tissue around a vessel with a dilatation of its adventitial lymph spaces, or it may be marked by a closure of the lymph spaces and a lymph stasis in the tissues from obstruction to the return flow of the lymph. Borst thought that a chronic inflammatory meningitic change initiates the process, leading to a closure of the epi-cerebral and epi-spinal spaces of His; the blood-vessel changes, especially around the para-central vessels, cause a closure of the adventitial lymph spaces, leading to a dilatation of the peri-vascular lymph spaces, and, with increased distension, the formation of cysts. As the congested lymph passes into the surrounding tissue, there comes about a hyperlymphosis with the formation of "Lichtungsbezirke." The obstruction to the outflow of the cerebro-spinal fluid by the obliteration of the adventitial lymph spaces and the closure of the epi-cerebral and epi-spinal spaces are therefore the causes of the disturbed lymph circulation. The acute infection which precedes the disease is stated to give rise to chronic inflammatory and proliferative processes in the meninges and vessel walls. In dependence on the lymph stasis a swelling and degeneration of the nerve fibres in the "Lichtungsbezirke" is brought about, and a later compensatory growth of neuroglia. The present writer's views in regard to the changes in the lymphatics and meninges have been stated elsewhere, and it is necessary here only to note that in these observations, in uncomplicated cases, no alteration in the meninges to which any significance could be attached could be found. The chronic

inflammation in the vessel walls, with a closure of the lymph spaces, is to be regarded as entirely a late change, so that while I agree with Borst that the fundamental basis of the process underlying disseminated sclerosis may probably be a flooding of the tissues with toxic lymph, I disagree with him regarding the mechanism by which this is brought about in the early stages. In late stages the condensed vessel wall, with adhesion of the adventitial spaces, on its side may contribute to the production of a vicious circle in which hyperlymphosis plays a part.

It is thus seen that I am in more or less disagreement with all the views related to the primary nature of the vascular lesions.

(3) THE ETIOLOGICAL FACTORS.

In turning now from the nature and origin of the process to its determining cause, we find that there is no positive knowledge of the nature of the agent causing this disease. The etiological factors postulated may be discussed under two groups: developmental and external.

1. *Developmental.*

In the discussion of the pathogenesis it was shown that Müller based his view overwhelmingly on the presence of an endogenous (developmental) process, under which naturally lay endogenous factors — inborn defects. One weak point in his arguments, however, lies in the fact that the evidence of congenital anomalies in the anamnesis is hard to find, or completely absent. Groups of cases occurring in families also have only been reported in one or two rare instances, and in this connection it must be remembered how great is the difficulty in the differential diagnosis between disseminated sclerosis and certain atypical forms of hereditary disease, and, further, that such cases might be family diseases with a symptom-complex like that of disseminated sclerosis. Again, cases of congenital multiple sclerosis are even more isolated. A neuropathic disposition has been alleged as an important etiological factor, but this association has not been substantiated, and where present no causal significance has usually been ascribed to it. Most observers note that patients were normal both physically and mentally.

Müller, in answering this argument of the absence of family

forms and neuropathic stigmata, points out that syringomyelia and glioma, attributed by numerous writers to congenital disturbances of development, have also no hereditary forms, and that heredity in nervous disease is seen chiefly in disease of the true nervous elements rather than in congenital anomalies of the glia. Nonne, who has examined the phenomena of inhibition of cobra poison haemolysis in organic diseases of the central nervous system, states that this cobra reaction is specially frequent in the psychoses which are claimed as types of endogenous etiology. As he has found it present in disseminated sclerosis more frequently than in any other organic disease of the central nervous system, he claims that this reaction supports the view of the endogenous nature of the disease. Nonne himself, however, gives indications in his paper that this result must be accepted with great reserve, for the reaction is found in general paralysis, admittedly an acquired disease.

Fürstner and others speak of "an early invalidity of the central nervous system," which is unloosed by later accidental factors. It is evident that such views do not bring the solution of the problem much nearer, yet it must be admitted that in some cases "congenital anomalies of development may be present in the sense that they lay the foundation of the constitutional tendency which renders the individual more susceptible to the injurious influences of later life" (Mott). Such anomalies may be insufficient to give rise to any symptoms till the onset of these later factors, but they indicate a diminished resistance or a diminished vitality of the nerve elements such as Gowers postulated for Friedreich's ataxia ("abiotrophy").

2. *External Factors.*

Among the more immediate factors must be considered the following: trauma (physical shock), psychical shocks, chills, infections, and intoxications. Modern conceptions of disease do not admit of primary importance being attached to the influence of cold, injury, strain, and emotion in the production of diseases of the nervous system, but such influences, recent or remote, are repeatedly brought into relation to the onset of such diseases. As a rule we cannot go beyond the *post hoc ergo propter hoc* argument, for there are no other data available, yet it is necessary to consider briefly whether the histological data throw any light

upon the mode of action of such influences, which are at least admitted to bring about a lowered resistance of the nervous system to the causal agent.

(a) *Trauma*.—Slight degrees of dystocic lesions of the infantile spinal cord may lead frequently to slight degrees of hæmorrhage, and it is thought not improbable that these may be the basis of the diseases of the spinal cord which are related to the formation of multiple areas of sclerosis, or to the formation of cavities and later syringomyelia. Mendel is one of the principal advocates of the traumatic etiology of disseminated sclerosis. He believes that as a result of the trauma an impulse of movement is transmitted to the cerebro-spinal fluid, that this leads to pressure effects in the most delicate channels of both brain and spinal cord, and that through these alterations of pressure there may occur minute ruptures (of capillaries) and hæmorrhages. From the assumption that small hæmorrhages are sufficient to evoke symptoms of disease, there justifiably follows, therefore, the view that in the case of cerebral and spinal "commotio," intra-medullary hæmorrhages are responsible for the symptoms. That the symptoms frequently pass away rapidly must be ascribed to a rapid resorption of the poured-out blood, but Schmaus argues from the fact that, since permanent disturbances frequently remain after shock, processes of degeneration in the nerve elements, which are beyond our present methods of recognition, have occurred. Hence his expression "molecular alteration of the nerve elements." The slighter degrees, he thinks, lead to transient symptoms, the severer to focal areas of degeneration, which thus have their rise in a molecular alteration resulting from "commotio."

The assumption of a molecular alteration of the nerve elements is hypothetical, since it does not rest on demonstrated microscopic findings at the time, but in the opinion of many writers intra-medullary hæmorrhages or outpourings of lymph into the tissue, of traumatic origin, may be followed by swelling and degeneration which lay the foundation of a *locus minoris resistentiæ* for toxic infective agents. It is also thought possible that in such damaged areas, still capable of functioning, the damage may be completed by any other factors which lower the resistance of the tissue. In relation to multiple hæmorrhages, it is relevant to mention the view put forward by Taylor and others, that at least a portion of the spinal cord degeneration found in some cases of pernicious

anæmia is due to primary focal degenerations caused by hæmorrhages similar to those which occur in the retina and elsewhere in this disease. In the literature on disseminated sclerosis there are several cases reported in which the association between trauma and the onset of the disease was a very close and striking one, but, as a rule, the association was questionable, and frequently some slight disturbance, such as giddiness, the symptom of an already existing disease, was the cause of the accident. Wohlwill requires the following conditions before there is proof of a causal connection between disseminated sclerosis and trauma: (1) that the individual was previously healthy; (2) a not too short and not too long interval between the accident and the first symptoms; and (3) a somewhat severe trauma from which one might expect injury of the brain or cord.

It has been already noted that in this investigation there was no evidence for the existence of capillary hæmorrhages, except those probably pre-agonal ones, due to respiratory difficulties. There are few positive data for the further fate of spinal hæmorrhages, and from those which we have as to the length of time taken in the absorption of cerebral hæmorrhages it is necessary to be cautious as to the conditions in the cord. The remains of such may continue to be evident for a very long time—often with no sign of degeneration or softening around them.

(b) *Psychical Shock*.—The influence of severe terror and other marked emotions has often been related not only to the onset of the disease, but to a relapse or the aggravation of a present condition. A possible explanation may be found in the sudden changes of the circulatory conditions in the nervous system occasioned by such influences as act specially on the vaso-motor system. The profound influence of the vaso-motor system in the production of disease has scarcely been recognised, and in a later section its possible mode of action in disseminated sclerosis will be considered.

(c) *Chills*.—Krafft-Ebing has attributed considerable etiological significance to chills. In forty out of a hundred cases he traced this factor, and represents the relationship between the two in the contraction of the vessels favouring ischæmic degeneration of nerve fibres in localised areas—especially if collateral anastomoses were absent.

In regard to cold, fatigue, and psychical influences, it is

probable that these act as immediate factors by lowering the vitality or the resistance of the individual or in allowing, through vaso-motor influences, the emergence of a toxin already circulating in the blood. In the case of trauma, the possibility of the actual rupture of capillaries or of lymphatics must be admitted, and the consequent hæmorrhages or the toxic lymph thus admitted in sufficient concentration into the tissues will effect the primary degeneration of the nerve fibres and a secondary proliferation of glia.

(d) *Exogenous Intoxications*.—Oppenheim has laid special stress on the influence of lead, copper, and zinc, also of alcohol and carbonic oxide, and has met with such causes in eleven out of twenty-eight cases of disseminated sclerosis. He speaks also of a toxi-pathic tendency inherited by the children of workers in lead and other metallic poisons. Hoffmann, however, in an analysis of a hundred cases, found lead present as a factor in only one patient. It is evident that some more general causal agent must be at work, although such intoxications may be important as predisposing factors in the production of a toxic arteritis.

(e) *Infections and Endogenous Intoxications*.—Toxic-infective agents have long been held of primary importance. Pierre Marie has urged the recognition of acute infective disease as the actual final cause of disseminated sclerosis, which he believes arises on the basis of multiple inflammatory vessel alterations brought about by the infective agents of scarlet fever, measles, diphtheria, small-pox, puerperal fever, pneumonia, erysipelas, typhoid fever, cholera, &c., in combination with the organisms which produce a mixed infection. Many other writers have favoured the view of the relation of acute infectious disease to disseminated sclerosis, but Hoffmann has strongly opposed it, and Krafft-Ebing in an analysis of a hundred cases found this factor in the anamnesis only six times, and no organisms that can have any causal relationship to the disease have ever been identified, either in the blood the cerebro-spinal fluid, or the tissues.

Malaria.—Several cases of disseminated affection of the central nervous system in consequence of malarial infection have been reported, chiefly by Spiller and by Italian writers. These arise probably on the basis of a mechanical closure of the cerebral and spinal vessels by the parasites which form solid thrombi. This merely shows that malaria may be followed by a disseminated

disease of the central nervous system, and such cases tend to recover more surely than cases of true disseminated sclerosis.

Tubercle and syphilis are both generally admitted to have no significance in the etiology. Syphilis may produce disseminated areas in the central nervous system, but the histological characters of these have, as a rule, nothing in common with those of disseminated sclerosis, in which disease also the reactions in the serum and in the cerebro-spinal fluid and the cytological examination of the latter are all negative.

Endogenous intoxications are of a very varied nature, and include not only the toxins engendered within the body by bacteria, but also auto-toxins caused through abnormal metabolic or assimilative processes and those the result of deficient or abnormal secretions. Systematic investigation of metabolic changes in disseminated sclerosis have not yet been undertaken, nor has any toxin of any nature been isolated either from the blood or the cerebro spinal fluid. Investigations of the bio-chemical changes, analogous to those found in para-syphilitic affections, have thrown little light on the disease, but investigations along this line have not been extensive.

It is the unsatisfactory and contradictory result of each of the above-mentioned factors that has led numerous writers to seek for one common cause which will explain the real nature of the disease. Klausner, in a careful analysis of a hundred and twenty-six cases of disseminated sclerosis, has come to the conclusion that few of the alleged causal factors are of any importance. Müller, from the large number of causes postulated, has drawn the inference that none of these can be the essential one, and therefore feels justified in falling back on his view of a developmental cause to which any of the other factors may be an exciting agent. François, also, believes that only this congenital degenerescence of the central nervous system can explain the characteristics of disseminated sclerosis, and that infectious disease, chill, trauma, &c., allows this to come into play. It has already been pointed out that the assumption of the developmental genesis of the disease is insufficiently founded, and such a conception must appear, in the light of modern pathological knowledge, untenable, and justifiable only when every other factor has been eliminated.

The essential problem, then, lies still before us: What is the

nature of the special stimulus which originated this process? In the attempt to answer this question it is important to remember that the causal factor must be in operation over long stretches of years, allowing remissions of the disease and relapses. It would, therefore, appear necessary to exclude those agents which actually attack the organism and then disappear rapidly. It is admitted by all, except the supporters of the developmental nature of the process, that the topographical distribution of the areas points to the blood-vessels or their lymphatic sheaths as the route of conveyance of this agent, and the assumption of an infection or intoxication harmonises with this relation to the blood-vessels.

In spite of the absence of any positive evidence for the presence of bacteria the organismal cause of disseminated sclerosis cannot be excluded, especially in view of the conditions existing in syphilitic and para-syphilitic affections on the one hand and poliomyelitis on the other. It has just been stated that the clinical and anatomical picture requires that the agent must be one whose influence extends over many years. We know that in syphilis we have an actual living organism remaining present in the body for years, therefore the chronic and variable course of the disease in disseminated sclerosis is not against the assumption of an infectious genesis. But with none of the acute infectious diseases which have been considered by Pierre Marie as etiological factors can we assume that the organism is preserved in activity in the central nervous system, and the supposition that in consequence of these infective diseases mixed infections, with persistent micro-organisms, establish themselves rests on no sufficient proof.

In acute poliomyelitis the earliest changes described in the nervous system are hyperæmia, and a collection of small round cells in the peri-vascular spaces of the blood-vessels of the soft membranes: this peri-vascular cell infiltration flows along the vessels as they enter the cord and reaches its height in the grey matter around the branches of the central vessels. Together with this cell infiltration there is marked œdema and the presence of minute or extensive hæmorrhages. These three features are all dependent on inflammatory vascular changes, and may be regarded as the primary reaction of the nervous system to the virus of poliomyelitis. Again, as a result of the acute infective diseases, we may get an acute infective myelitis, and probably one portion of the cases ascribed to disseminated sclerosis, following

such conditions, must be ascribed to true acute myelitic conditions, for Pierre Marie regarded the primary participation of the blood-vessels as an anatomical proof of his clinical theory of the relationship. Cases of acute infective infiltrative myelitis, with organisms present in the foci, have been described by Purves Stewart and others, and there are also other cases in which it is impossible to state the exact nature of the infection. Further, the experimental investigations of Marinesco, Hoche, Homen, Salle, and others have corroborated the organismal cause of certain forms of myelitis by the production of focal areas of infiltrative myelitis as a result of the infection of micro-organisms. These writers believe that organisms exert their action primarily upon the wall of the blood-vessel.

The forms of myelitis have been classified by Taylor and Buzzard as infective, toxic, and syphilitic. The last-named variety shows changes which we have already noted as being readily recognised, and as quite distinct from the changes in disseminated sclerosis, so that it need not be further considered. The experimental work we have above referred to has shown that areas of infective and toxic myelitis may be produced by a number of bacteria and bacterial toxins, and a comparison of the histological changes in the two forms with those of similar conditions in man gives the following as the general differential histological characteristics. In infective myelitis we have dilated and engorged vessels, with an excess of nuclear elements in the adventitial sheath, probably derived from the proliferation of the structural elements of the adventitia rather than from the blood (Taylor and Buzzard). Later there is proliferation of the glia elements and changes in the nerve cells and fibres, partly due to oedema and to the vascular changes, and partly to the toxic products of the invading organisms. In toxic myelitis, on the other hand, the changes are much less marked: the patchy areas of degeneration are unaccompanied at first by any cell infiltration of the vessel-adventitia or of the tissue, and there is, as a rule, marked swelling and varicosity of the axis cylinder and myelin sheath. The later reactive changes in the glia depend on the intensity of the primary change.

From the evidence of an infiltrative poliomyelitis, caused by the virus of poliomyelitis; from the recorded cases of acute infiltrative myelitis in man; and from the experimental production

of infiltrative myelitis by bacterial injection into animals, it is natural to assume that organismal infection is more often associated with primary inflammatory (proliferative) changes in the blood-vessel walls. Taylor and Buzzard, however, point out that some organisms may at one time and under certain circumstances determine an infiltrative form of myelitis and at other times a toxic, and that we are not fully acquainted with the laws which govern such different results. It must also not be forgotten that toxins circulating in the blood or lymphatics may also produce lesions first in the vessel walls; the work of Orr and Rows has definitely proved that a cellular reaction in the adventitia may be due to a chronic lymphatic intoxication—the presence of numerous plasma cells in this reaction is the expression probably of a chronic inflammatory process. The balance of evidence, however, goes to prove that an infiltrative myelitis is usually due to an organismal cause, and most workers conclude that areas of necrosis, without cell infiltration and independent of vascular thrombosis, must be ascribed to toxic influences. The toxic myelitis of pregnancy may be given as an evidence of the result of an auto-intoxication which is often responsible at the same time for serious trouble in connection with the cardiac and renal functions.

Turning now to the histological data previously given, it will be recalled that the conviction has been expressed that nowhere was there evidence of a primary vascular inflammation, but that the first evidence of cell increase in the adventitia was an infiltration of fat granule cells consequent to resorptive processes, that the subsequent proliferation of adventitial cell elements was secondary to and probably caused by this infiltration, and that only at a still later stage was there an infiltration of small lymphocyte-like cells analogous to those found in all chronic processes. It has been further stated that the histological observations lead to the conclusion that the stimulus acts primarily on the myelin, and almost simultaneously on the glia cells, surrounding the blood-vessels. The changes which have been outlined in a normally developing "early" area are thus much more nearly allied to those of a toxic myelitis than those of an infective myelitis, and differ from the former only in degree.

In the absence, however, of any definite proof of such a toxin either in the blood or cerebro-spinal fluid, other evidence must be submitted with great reserve. The example of general paralysis,

in which disease Noguchi has demonstrated the presence of the *Spirochæta pallida* in the cortex, but which formerly was ascribed to a chronic toxic encephalitis of lymphatic origin, must serve to show how quickly views regarding the pathogenesis and etiology of a disease may require to be modified. In this instance, however, it may be stated that the most recent writers on this subject, Mcintosh and Fildes, look upon the inflammatory reactions—perivascular infiltration of lymphocytes and plasma cells involving the vessels of the pia and those that penetrate the cerebral substance; “primary degeneration” of the neurons; and proliferation of the glia—as all due to a primary reaction to the *Spirochæta pallida* and not primary or secondary to one another, while Nonne (quoted by those writers) looks upon the lesions not as the direct result of the activity of the *Spirochæta pallida* but as due to the action of the toxins, derived from them, which have a special affinity for nerve areas.

The important point, however, in relation to disseminated sclerosis is that experimental work has proved that circumscribed areas may be attacked by toxic materials, and that in such areas the first effect is on the nerve fibres and interstitial tissue surrounding the blood-vessels and only secondarily on the vessel walls themselves. The causal agent must be a weak one compared to the usual agents which produce toxic myelitis, for there is evidence of a slow, often limited and localised action, and the subacute onset in the relapses again argues for its slight intensity. For such a chronic course as disseminated sclerosis usually runs, with many remissions, the “noxa” responsible must remain constant in the body, lead at certain periods to relapse, or even remain latent for a long period. The assumption of an auto-toxin agrees with this demand, and the example of pernicious anæmia in which the remissions may probably be traced to the result of absorption into the portal tract of some poisonous substances may serve as a possible analogy. The assumption of an organismal stimulus moving about in the body or malignantly stored in depots such as the spleen or bone-marrow, or of the elaboration products of such organisms continually reforming and leading to new symptoms, would also agree with this demand. It is impossible to say that this is improbable, for the relapses in malaria and relapsing fever coincide with the reappearance in the circulating blood of free sporocytes of the malarial organism or of the spirillum of Obermeyer,

respectively: such relapse is therefore a re-infection from within (Ainley Walker). In para-syphilis, also, it must be supposed that the specific organism in some form enters the central nervous system, remaining latent for years either in the lymph vessels or in the brain tissues proper, and exerting its effect either by its direct action or by its toxins. The influence of such factors as infectious diseases, cold, trauma, &c., would thus be to produce an impairment of the nervous tissues, and so predispose them to degeneration from auto-toxins and other causes: or the lowered body resistance would allow of sufficient concentration of the toxin, through deficient elimination: or a combination of both factors, impairment of the tissues and an increasing intensity of the "noxa," probably acts in varying degrees in every case.

The views that have been put forward as to the source of the possible "auto-toxin" can be only briefly touched upon. M'Cormac suggests that disseminated sclerosis is due to physico-chemical causes, which may be in operation for a long time, and thinks that as indol, skatol, and phenol—toxic products of intestinal putrefaction—might produce structural changes in the liver which are followed by nervous systems, it is possible that actual structural changes might occur in the nervous system itself. Bramwell suggests several possibilities: the toxic agent may be the result of deranged metabolism, possibly in the liver or some other organ, or it may be generated in and absorbed from the gastro-intestinal tract, as is probable in pernicious anæmia. The effect of exposure to cold in the production of hæmoglobinuria is explained by the probable rapid production of some toxic substance, the result of deranged metabolism. It is thought that the exposure to cold acts reflexly through the nervous system on some central organ—perhaps the liver—and produces a poison which is the cause of the rapid destruction of red blood cells, and that disseminated sclerosis might be the result of a chemical poison similarly formed. Another possibility is that the agent may not be a toxin properly so-called, but that the composition of the blood is so altered that some substance or substances necessary for the nutrition of the nervous tissues, especially the nerve fibres, is absent, or that the blood contains some substance which acts injuriously on the nutrition of the nerve fibres. Dixon Mann thinks that disseminated sclerosis is due to an unknown autogenous toxin, which probably acts by setting up changes in the ultimate vascular supply of the part affected.

(4) THE MODE OF ACTION OF THE CAUSAL AGENT.

It must have been noted that as this chapter has progressed we have got deeper into the realm of theory. In discussing the nature and the origin of the process there were certain definite, though restricted, data to go upon: in regard to the final causal factor there were few data, but the hypothesis of a latent organism or a circulating toxin rested on certain analogies: and when we come to discuss the mode of action of this causal agent, and the further questions which this consideration presents, it is seen that in relation to some at least of these questions recourse has been taken still more to analogy. Yet it is necessary, at least, to state what these questions are, and, at most, to indicate briefly any reasonable explanation that has been put forward to answer them.

The principal questions which present themselves in this section may be stated thus:—

1. Why and how is the process so irregularly distributed and at first circumscribed?
2. What is the cause of its further advance?
3. What factors cause modifications in its mode of action?
4. By what route, blood or lymph channels, is it conveyed to the nervous tissues?

1. *Its Irregular Distribution and Circumscription.*

If we could trace the disease to the entry of corpuscular elements, such as bacteria, or thrombi infected by such, or emboli, there would be no further difficulty in accounting for circumscribed vascular areas being irregularly attacked. If it is not, however, a question of the immediate action of bacteria at an identical point, but probably of toxins circulating in the blood, how is it that these agents in solution in the blood choose such irregularly distributed spots?

From the frequent presence of a central larger vessel in the area it has been assumed that this vessel, by some alteration in its walls, limited to a certain definite stretch of its longitudinal course, was responsible for the sclerotic area. Strümpell and Müller, who opposed the view of the rôle ascribed to the vessels in the process, naturally found in this an argument in their favour, for they found it difficult to perceive how toxins could

diffuse from such a large vessel instead of following the capillary area of ramification. It seems to me that Müller has missed the significance of the vessels largely because he has preferred the cord areas which do not allow these relations to be so readily recognised, but, on the other hand, it is probable, as has been frequently shown in the histological study, that it is not a question of one central vessel but of many transverse and oblique vessels, giving the impression that not one single vessel, but the branches of a blood-vessel stem are the starting-point of the process. Serial sections have frequently given definite proof, as the area was followed up, that it broke up into individually distinct smaller areas, which were related to the component branches of the previous central vessel. These sections, again, showed the gradual fusion of such areas around the ramifications of a vessel, and marginal strands of myelinated fibres still left between each primary focus. It must not be forgotten, however, that the whole vessel territory need not be attacked, and, on the other hand, that the area of ramifications of non end-arteries has no sharp definition. The peri-ventricular areas, which we trace to the involvement of the terminal branches of the central arteries, which spread out on the ventricular surface, are a striking illustration of the implication of the area of ramification of end-arteries, and an instance equally marked is the demyelination of the surface layers of the cortex corresponding to the area of supply of the smaller cortical vessels.

The difficulty of understanding how a "noxa," *e.g.*, a circulating toxin, in the circulation could assert itself in the immediate area of a large vessel, for a limited distance—so far as it had not to do with an actual disease of the vessel wall itself—rather than in the region of the capillaries which are distributed everywhere, has led numerous writers to fall back upon the assumption of a special predisposition of these areas either in the form of congenital defects or acquired *loci minoris resistentiæ*. Such a predisposition was also held to explain the frequent symmetry of the areas, but this is again more readily understood if we admit as the histological evidence favours, that it is the area of distribution of the vessels, which may naturally be of very varying extent, that is affected. Such a view, however, removes only a portion of the difficulty. If certain areas of distribution, large or small, are affected, what factors determine the exact areas chosen?

For this no satisfactory explanation has been suggested, but certain analogies may be given.

In peri-axial neuritis in man, the result of chronic lead poisoning, not only is the radial nerve picked out, but certain discontinuous areas are primarily affected in a manner very closely resembling the discontinuous myelin sheath degeneration in disseminated sclerosis. Gombault and Stransky have also experimentally produced similar discontinuous areas in subacute lead poisoning in animals. In these cases the toxin circulating in the blood is known to affect only certain irregularly distributed areas. The selective action of toxi-infective agents on particular parts of the nervous system may be further illustrated by the action of the virus of syphilis and that of rabies; by the effect of arsenic and that of the neurotoxin formed by the diphtheria bacillus, on certain nerves; and further, by the tetanus toxin, which like strychnine, acts mainly on the synapses of the reflex arcs of the brain stem and spinal cord, reducing their resistance.

Further, the combined degenerations found in pernicious anaemia, cancerous cachexia, diabetes, and the sclerosis found in chronic ergot poisoning, pellagra beri-beri, and other conditions, pick out the posterior columns or both posterior columns and the posterior part of the lateral columns. In pernicious anæmia, at least, Minnich and Nonne have traced such degenerations to primary focal areas in the posterior or lateral columns: such areas, later, either from the cumulative effect of several foci at different levels in relation to the same strands of fibres, or from the later involvement of the axis cylinders, are succeeded by secondary degeneration. In such conditions the toxin postulated must choose out certain definite vessel territories, and the symmetry is explained by the two halves of the cord being exposed to the same diffusely acting agent. Numerous writers, however, suggest that prolonged toxæmic states lead to primary degeneration of those parts of the cord which are apparently more sensitive to trophic disturbances, and either that the posterior and lateral columns have a poorer vascular supply or that, as the parts most affected are the neuraxons of the posterior spinal ganglia and those of the cortical motor cells, therefore the long terminal filaments of the cervical and dorsal cord, they are far removed from the influence of their trophic cells. When it is urged that certain areas of the nervous system may have a "general non-

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resisting power," it can be understood how such functional factors as overstrain may affect definite strands and areas, related to definite functions, but this view, however unsatisfactory and unconvincing even for such conditions as cause combined column degeneration, carries no weight when related to areas with the haphazard distribution found in disseminated sclerosis, for one of the characteristics of the disease is that the areas bear no relation to any functioning tract or group of cells.

The sequence of events in disseminated sclerosis is probably that in the circulating blood the "noxa" escapes from the capillaries and transition vessels, and acts upon the tissues in the sense that the area of supply of the vessel is affected, *i.e.*, it passes over into the tissues with the nutritive fluid. Numerous recent experiments on man and animals tend to prove that, in general, drugs administered by the mouth or subcutaneously do not pass into the cerebro-spinal fluid, and Mott has pointed out the significance of Lewandowsky's observations, which show that very much smaller quantities of these same drugs and bacterial toxins injected into the cerebro-spinal fluid of the sub-arachnoid space produce much more marked and a more rapid onset of symptoms. These and other observations are taken to prove that toxic substances are unable to pass from the capillaries into the lymph spaces of the nervous tissue. Goldmann's investigations on the central nervous system by vital staining show that vital stains, if introduced by means of subcutaneous or intravenous injection, are kept back by the choroid plexus: that from the plexus the cerebro-spinal fluid receives important metabolic products which are carried to the nervous tissues by the fluid; and that the plexus possesses the power of protecting the fluid, and in this way the nervous substance, from the penetration of toxic substances. The rôle of toxins in the production of nervous disorders has long been recognised, and also the rôle ascribed to the secretions of the protective organs of the body—amongst which must now be enrolled the choroid plexus—in neutralising the products of auto-intoxication. A defective function of these organs will cause an accumulation of such toxins in the body, which will immediately react on the nervous system. Orr has shown how such a primary intoxication may so lower the resisting power that micro-organisms and their toxins may gain access to the blood stream.

Unless we admit, however, that such substances circulating in the blood can pass from the capillaries, how can we explain the action of poisons on the nervous system? Williamson attributes considerable importance to the cell walls of the capillaries and lymphatics of the central nervous system in reference to the pathology of disseminated sclerosis. He believes that the cells of the capillary walls act as true secreting cells, and when they are stimulated there is an increased flow of lymph into the surrounding tissue. Certain substances, such as toxins produced by micro-organisms, have the power of stimulating the endothelial walls, and as the endothelial walls of the central nervous system of capillaries and lymphatics are extremely delicate and active, the lymph flow is great. Numerous writers suggest that the toxins produce an unrecognisable injury of the vessel wall by which it becomes more permeable, and thus allows of the increased transudation of the lymph. Others think it possible that a transient paralytic dilatation of the vessels permits an exudation, which in other conditions does not pass through the vessel wall or is arrested by the glia filter. The conditions of the cerebral circulation are so little understood, and the vaso-motor mechanism so complicated, that such an irregular and localised vaso-motor action may be quite possible. The presence of large areas of demyelination, such as those seen in Case II., in which every level of the cord showed an almost complete transection, gave the impression, however, that such an extensive demyelination is preceded by acute vascular dilatation, and that the tissues are flooded with toxic lymph which has caused a rapid solution of the myelin. The dilated vessels found in all the "early" areas might also argue in favour of a persistent paralytic dilatation from want of vascular tone in the area.

None of these suggestions explain why certain areas are chosen while adjoining areas of distribution are, at least primarily, unaffected, and it may be that its causation has appeared unnecessarily mysterious and that it is quite analogous to the irregular distribution of other processes, *e.g.*, the haphazard distribution of areas in experimental toxic myelitis or the atheromatous patches on vessel walls. It cannot be supposed that the tissues are in a state of proportionate equilibrium or equal resistance, and that there are not varying degrees of oscillation and pressure and permeability in the vessels. The filtration from

the vessels, including whatever part secretory processes may play in the passage of the lymph through the vessel walls, may thus be related to alterations, the nature of which are as yet little understood, but which have a place in the normal mechanism of the body.

The frequent sharp delimitation of the primary isolated areas gives the impression that the spread of the process is in the nature of the diffusion of a toxic substance which spreads from a central focus till it exhausts itself. Williamson puts forward a very suggestive view of the causation of this characteristic. The margin or outline of an area is noted as passing through the structure of the brain or cord substance regardless of fibre tracts, nerve cells, or vessels. He suggests that this is due to the infiltration of the nerve tissue with a fluid of destructive character, the shape and margins of the area being determined by physical conditions, since patches of similar form can be produced when stained fluid is allowed to infiltrate the cord (post mortem) from various points.

2. Further Advance of the Process.

The ebb and flow of the symptoms are, clinically, amongst the most characteristic features of the disease, and, anatomically, "early" areas are the expression of this recurrence of a long-standing disease. When the primary direct effect of the causal agent has become exhausted the areas become sclerosed, and in such cases there may be a true remission, but frequently in addition to "early" areas there are found at the periphery of the old areas indications of an advancing process. This may be due to the persistence of the action of the primary agent, or to the fact that the products of degeneration had caused a secondary reaction after the infective or toxic agent had ceased to work, or, possibly, to a new process surrounding the older one. Such recent areas show that the morbid agent persists in the organism. Müller has urged that the similarity of the basal features of the clinical and anatomical pictures points to one common cause of the disease: he thinks that we cannot assume that, for example, the toxo-infective agents of the acute infectious diseases, *i.e.*, of so different a nature, will always lead to one and the same definitely characteristic disease-picture. A similar view has been upheld by numerous writers, and, in the absence of any specific organismal

or toxic cause, an explanation has been sought for along very varied lines. Redlich thinks it possible that the characteristic symptom-complex is caused by the "general functional injury" to the nervous system: that the acute infectious disease, whose cause we otherwise look upon as specific, may yet produce a definite metabolic disturbance, which, in its action upon the central nervous system, is specific. Marbourg also looks upon disseminated sclerosis as a "meta-infectious" disease in this sense. Such a term seems justifiable in view of the use of the term meta- or para-syphilis in which it was believed that the syphilitic virus induced in the body profound metabolic changes. Mott has pointed out that these resulted in a large amount of lipoids occurring in the serum and in the cerebro-spinal fluid: that these same lipoids are found in the normal body fluids, so that the specific character is thus manifested by quantity rather than by quality. The relations of syphilis to the para-syphilitic affections are of special interest in relation to the possibility that a latent organism may be the cause of disseminated sclerosis. Before Noguchi's discovery of the *Spirochæta pallida* in the cortex, Mott and Sewell had defined these relations thus: (1) the syphilitic organism is in itself the causal organism; (2) there is produced within the body as the result of syphilis a toxin, and this post-syphilitic toxin is the cause; or (3) syphilis produces an impairment of the nervous system and so predisposes it to degeneration from auto-toxins and other causes. It is in this final sense we look upon Redlich's and Marburg's conception—that acute infectious diseases of very varied etiology may produce once for all a specific impairment of the nervous system, and so predispose it to degeneration from auto-toxins. It has also been suggested that the original agent may cause, in addition to the evident areas of degeneration, alterations in other portions, and that these later, not from a persistence in the organism of the causal agent, but from excess of function, strain, or circulatory disturbances, may degenerate. Again, that the presence of degenerative processes in the central nervous system may suffice in themselves for the production of further areas of degeneration. For either of these views there is no foundation nor analogy. In the absence, therefore, of any positive evidence as to the determining factor, it can only be presumed that relapses are due either to the intermittent evolution of a toxin, or to an unknown organism

which, in the interval between a remission and a relapse, lies latent and inactive. The causation of pernicious anæmia, as we have seen, is thought to be due to the similar intermittent evolution of a toxin, and the discovery of the *Spirochæta pallida* in the cortex in general paralysis is a sufficient analogy for the latter alternative. Mott has suggested that this organism may exist in a latent, granular, or intra-cellular form in the parenchyma of the nervous system, where it cannot be reached by drugs such as arsenic, mercury, and antimony.

3. *The Factors which cause a Modification in the Action of the Causal Agent and the Relation of the Process to Acute and Chronic Myelitis.*

The variations in the histological picture have been ascribed by different writers to two main causes: the fact that the process was observed at different stages, and to the different nature of the etiological factor in individual cases. Apart from such basal differences and from the variations due to the influence of complications, there are modifications in the development of the process which can be attributed only to the fact that its progress, assuming one common causal factor, is not always typical, uniform, and progressive.

Ainsley Walker, in discussing the relative changes in the tissue elements in inflamed areas, points out that stimulation and injurious irritation differ only in degree, and that whatever is capable of causing injury will, if sufficiently diminished in intensity, exert a stimulant action. On the other hand, stimulation, if sufficiently intensified or long maintained, becomes irritation and produces injury. He further points out that one and the same agency may have a different effect on different types of cells, or even on different cells of the same type. The changes which occur in the tissues of inflamed areas are, therefore, of two types, degenerative and regenerative respectively. Both of these occur simultaneously in different orders of cells, *e.g.*, the parenchyma of an organ may undergo degenerative change while its connective-tissue basis is proliferating. If we apply this to the changes in disseminated sclerosis, we can conclude that "there are certain principles underlying the inflammatory process which enable us to recognise in it different degrees of one process

rather than several independent series of reactions." This shows that a common cause, according to the intensity and duration of its action, may produce a very varying picture.

If we assume as the causal agent in disseminated sclerosis a circulating toxin, it has been stated previously that this toxin must be in such weak concentration that it produces no recognisable injury on the vessel wall in passing through it. This weak toxin is further assumed to have an affinity for myelin, and it produces, in the immediate neighbourhood of the vessel from which it has passed out, a simple primary degeneration or solution of the myelin, with a proportionate reaction on the glia—thus its injurious action is exercised on the myelin sheath, its stimulant action on the glia. As this diffusion extends and the toxin mixes with the tissue fluid and becomes more dilute, its stimulant action would be more in evidence, and when the toxin tends to exhaust itself, *i.e.*, at the peripheral zone of the primary area, there would be a solely stimulant action on the glia, an action which extends, therefore, beyond the area of degeneration of the myelin. This stimulant action of the toxin seems to us to account for two of the histological data brought forward by Müller in evidence of a primary glia change: (1) that at the periphery of the area we have a glia nuclear proliferation; and (2) that this extends between the normal myelinated fibres at the margin of the area. These two data may possibly be partly explained by the secondary glia proliferation occasioned by the degeneration of the nerve fibres, but for our present purpose it is necessary to emphasise the stimulant action of the primary causal factor on the glia—a stimulus which increases according to its dilution. The development of such an area is that pointed out in tracing the evolution of an area through a stage of fat granule cell myelitis.

If we assume, further, that the concentration of the toxin is still more dilute from the commencement, the degenerative action on the myelin would be almost in abeyance, and the stimulant action on the glia would be its sole effect. A slowly-increasing glia hyperplasia could then result, which would lead secondarily to a myelin degeneration partly by direct compression and chiefly by the alterations in the blood and lymph circulation in the area. Such a stimulus would thus lead to an area of sclerosis through stages of a "gradually increasing glia hyperplasia," which we have previously outlined. If such an assump-

tion is justifiable, the two types of areas are not two individually distinct processes, the latter developmental and the former inflammatory, but both are occasioned by the same causal agent acting with different intensity and over a longer time. Such a slowly increasing interstitial change is, therefore, an illustration in the central nervous system of the fact that the first evidence of reaction, when the action of an irritant is slow enough for us to follow clearly its results, is a proliferative change.

If we assume still further that the toxin is in greater concentration and acting more quickly, the first effect would be solely on the myelin sheath, which would degenerate rapidly. If this were not followed by a compensatory glia proliferation, there would result the type of area described by numerous writers as "areolierte" areas, in which the myelin sheath is dissolved away, leaving the original network of the glia and the axis cylinders persisting. If there were an attempt at a substitution glia proliferation, the glia nuclei would form the nodal points of a brush-like formation of fibrils, but this fibril formation would yet be insufficient to fill up all the meshes of the tissue, and the resultant area would be midway in its sclerosis between an "areolierte" area and a dense sclerotic area. Both of these latter types of areas are very numerous, and give the justification for looking upon the change in the myelin sheath as the most constant and uniform one.

It is possible to go a stage further and assume that the toxin is so concentrated, or acts so rapidly, that it attacks not only the myelin sheath, but destroys the axis cylinders and goes on to destroy the meshes of the glia, which thus break into one another, giving the appearance of the so-called "Luckenfelder." We would thus get true myelitic areas, and this is the present writer's view of the relation of disseminated sclerosis to acute myelitis. The areas in disseminated sclerosis are areas of lesser degeneration, and the difference in the pathological process is one only of degree. I assume, therefore, that the changes are not so intense in degree as in acute myelitis, and I regard disseminated sclerosis as a localised disseminated subacute inflammation, which gradually tends to sclerosis. In such subacute processes the general architecture of the tissue is retained. Chronic myelitis is a term which is becoming obsolete. It can represent the remaining stage of a previous acute myelitis, of a process

which has begun chronically, or is healing slowly. Taylor and Buzzard state that disseminated sclerosis has some claim to be regarded as a chronic inflammatory disease of the spinal cord, and is sometimes held to be the only true instance of chronic myelitis.

It is difficult to answer the question whether an acute encephalo-myelitis can pass over into a disseminated sclerosis. The pathological conceptions of the disease, as have been indicated, pass over into one another, and with this conception is admitted the inflammatory nature of the process. Whether it can be regarded as inflammation, as degenerative inflammation, or as purely degenerative, depends fundamentally upon different definitions of the same process. It seems defensible to regard certain forms of disseminated sclerosis, especially those occurring in close relation to the acute infectious diseases, as having their origin in an acute disseminated encephalo-myelitis, but, in spite of the seeming inconstancy of the symptoms and the irregular incidence in the position of the areas, it seems justifiable to regard disseminated sclerosis as an inflammatory process with a subacute onset and slow progressive course—often distinguished by remissions and acute or subacute relapses which depend upon the development of new areas, and to regard the final cause as a true specific “noxa,” which may be either a metabolic disturbance or a special infective stimulus.

4. Route of Conveyance of the Causal Agent to the Tissues.

Writers admit two paths of infection of the central nervous system—the one, which has been looked on as the more constant—the blood stream, and the other the lymph stream. The possibility of lymphogenous infection of the nervous system has received much attention since experimental evidence was established in favour of the spread of rabies and tetanus by the lymph channels of the nerves. Its increasing recognition in this country is largely due to the work of Orr and Rows, who have taken as the principle of their research the fact, demonstrated by numerous experiments with organisms and coloured fluids, that the lymph stream in peripheral nerves is an ascending one and capable of conveying infection to the central nervous system. The main current of this ascending lymph stream is said to lie in the inner

meshes of the peri-neural sheaths, and when it reaches the cord, chiefly by the posterior roots, it for the most part passes along the entering posterior nerve roots into the substance of the cord, and the remainder is distributed in the inner meshes of the arachnoid around the whole surface of the cord. The lymphatic path within the cord has, in the main, an outward direction, as is demonstrated by the presence of fat granule cells, containing the degenerated products of the nervous tissue within the adventitial spaces, but the experiments of Homen, Salle, and Marinesco, together with numerous histological observations, leave no doubt that it admits of a current inwards—thus admitting an invasion by cellular elements, micro-organisms and toxic substances.

In the experiments of Orr and Rows, celloidin capsules containing a broth culture of an organism was placed in contact with the sciatic nerve. The path of the toxic lymph could be traced by the inflammatory reaction in the sciatic nerve, posterior root ganglia, and along the spinal roots. If the capsules were placed near to the spinal cord, in order to lessen the distance along which the infection had to be conveyed, this reaction was evidenced within the cord substance itself, and its characteristics depend entirely on the potency of the irritant. When the capsules had not burst and the tissues were attacked by toxins only, the reaction was of a plasma-cell type, but when the capsules had burst and the organisms had grown in the tissues, there was an intense proliferation of cells of polyblast type. As the same animal was used and the same organism, the differing reaction must be attributed to the difference in quantity and potency of the irritant. The reaction in the spinal cord was produced in both instances by injected lymph which spread by the same path—along the adventitial lymph spaces of the vessels entering from the pia, and therefore attacked the same cells, those of the adventitial sheath of the vessels. The plasma cell is looked upon as the type of cell characteristic of subacute inflammation, and the polyblast as characteristic of an acute inflammation in the central nervous system. If the toxin or organism gaining entrance to the central nervous system by this source be weak, or penetrate the tissues slowly, no other phenomena but that of adventitial proliferation need occur for some time, and the changes in the cord diminish in degree from without inwards.

These experiments tend to prove that infection passing into

the cord by the lymphatic system takes a definite course, that the structures of the cord and the nerves react to infection by this path in a definite manner, and that the inflammation can be propagated by the toxic lymph to parts distant from the focus of greatest intensity. It is recognised that when once the inflammatory condition has been established within the spinal cord, the toxic lymph spreads by direct continuity, and this continuity of extension is looked upon as characteristic of lymphogenous infections. Numerous clinical data, from cases in which peripheral inflammatory foci existed, have been brought forward by Orr and Rows, in support of their experimental work, to demonstrate the facility with which infection spreads along the lymph sheaths of nerves to the spinal cord. Here also the histological changes in the membranes and nervous tissues showed that the reaction varies with the potency of the irritant, and that the degree of reaction in the nervous tissues diminishes from without inwards. In connection with these changes the cord infection which sometimes follows inflammation of the urinary bladder is referred to and is of interest in relation to the evidences of inflammatory changes in the membranes and in the peripheral vessels of the cord found in some of our cases. Orr and Rows further apply this principle of lymphogenous infection to acute polio-myelitis and general paralysis. They look upon the histological changes in the former disease as showing no essential differences to those found in their acute cases—in both the preponderating cell type is the polyblast. The changes in general paralysis also appear explicable only by the presence of toxins of organisms gaining access to the lymph which bathes the brain and membranes and circulates in the adventitial lymph spaces of the cortical vessels, calling forth a chronic peri-arteritis of a plasma-cell type.

In relation to acute polio-myelitis it may be stated that experimental evidence proves without doubt that it can be produced by lymphogenous infection, and the view that the virus may enter by means of the lymphatics and thus exert its first effect upon the meninges is strengthened by the anatomical findings (Peabody, Draper, and Dochez). The earliest change described in the nervous system is hyperæmia and the collection of numbers of small mononucleated cells in the peri-vascular lymph spaces of the blood-vessels of the lepto-meninges. The lymph spaces surrounding vessels of the cord are, anatomically, processes of the arachnoid

space, and the lymph in them is in connection with the cerebro-spinal fluid. With the advance of the pathological process this peri-vascular infiltration follows along the vessels as they enter from the meninges and is most marked around the central vessels. The evidence, first pointed out by Flexner, that the respiratory mucous membrane provides for both the ingress and egress of the virus, has much to support it. Romer and Wickman, on the other hand, on clinical and experimental grounds, think that the virus has its habitat in the intestinal tract, and thence finds its way along the lymphatic sheaths of the sympathetic nerves to the central nervous system. Once this is reached an infection of the lymph spaces in the adventitial sheath of the veins in the pia mater and spinal cord immediately follows. Wickman supports his view of the lymphogenous origin of acute polio-myelitis on the following histological grounds: that in many parts the chief and only change consisted in an infiltration of the larger vessels, while the capillary region of such vessels was quite free; that the changes in the longitudinal axis were continuous—a continuity which reaches its maximum intensity in Landry's paralysis, and that the infiltration of the adventitial sheath of the vessels argued for the causal agent circulating in the lymphatic spaces.

It has been assumed by most writers that the causal agent in disseminated sclerosis circulates in the blood, but the possible lymphogenous source of the infection has been supported on the following grounds: (1) the endo-vascular changes are much less marked than the peri-vascular; and (2) the pathogenic significance of the peri-ventricular sclerosis. With reference to the former argument, it is again necessary to point to the fact that in this investigation peri-arteritic changes, in the sense of adventitial nuclear proliferation, were completely absent in the early areas before the onset of a secondary cell infiltration due to resorptive processes had occurred. There was thus no evidence in the cell proliferation of the adventitial sheath that a "noxa" was circulating in the adventitial spaces. In view, however, of the findings of Orr and Rows, that in infection of the cord by the passage of toxins along the sympathetic nerves no adventitial proliferation was found, such a possible source of lymphogenous infection cannot be denied.

The peri-ventricular localisation was such a striking feature in several of the cases that early in the investigation it was recognised

that ependymal and peri-ependymal lesions lead to important considerations in reference to the toxicity of the cerebro-spinal fluid. Bullock's recent experiments point to this toxicity, and Lhermitte and Guccione found that when carmine was injected into the lateral ventricles of a dog, it was found, ten days later, almost wholly in the sheath of the sub-ependymal veins and in the sub-ependymal tissue. It is logical, therefore, to assume that toxi-infective agents in the cerebro-spinal fluid might follow the same route. The absence, however, of any change in the ependymal epithelium seemed to contraindicate the possibility of a simple soakage of a cerebro-spinal fluid into the peri-ventricular tissue and also to contraindicate the irritating character of the fluid, for a granular condition of the ventricular walls is usually associated with such a change in the fluid. Borst has pointed out that the terminal branches of the central arteries ramify on the ventricular walls, and this localisation of the areas might equally be related to the vascular richness of this region. The cell infiltration and proliferation around the sub-ependymal veins, which constitute the path of return of the peri-ventricular circulation, would then again be simply an indication of the resorptive processes consequent on myelin degeneration and not a result of a peri-ependymitis. Lhermitte and Guccione come to the conclusion that the toxic agent in disseminated sclerosis is carried mainly by the blood stream, but that a part in the process must also be ascribed to the cerebro-spinal fluid.

Extra Note.

The wide-reaching possibilities of lymphogenous infection in connection with the elucidation of the etiology of some nervous lesions is well illustrated in the recent work of Orr and Rows. These writers have kindly given the following account of this yet unpublished work. They consider that the lymphogenous infections are characterised by phenomena varying from a polymorpho-nuclear or polyblast cell exudation in the acute processes to a plasma-cell reaction in the chronic, and that peri-arteritis is an essential feature. On the other hand, when bacteria-laden capsules are placed in the abdominal cavity—a position least likely to lead to infection of the lymph sheath of the peripheral nerves—it is found that no peri-arteritic plasma-cell

formation occurs in the spinal cord. The changes so far observed have been as follows: (1) hyaline degeneration of the vessel walls with hyaline thrombosis; (2) neuroglia proliferation around the vessels; (3) the nerve cells are practically normal; (4) there is no evidence of peri-arteritis; (5) small areas of disseminated sclerosis; (6) a slight degree of myelin degeneration which varies in distribution in the different levels of the cord; (7) the sympathetic nerve cells in the abdominal chain show chromatolysis. They are of opinion that the influence of the sympathetic cannot be excluded, and that the view of a general intoxication cannot be sustained as an explanation of these lesions owing to their patchy character. They incline to the view that the involvement of the sympathetic mechanism here and there causes dilatation and stasis in certain parts of the cord vessels, favours the formation of hyaline thrombosis, and hence myelin atrophy and sclerotic areas. The work on which these guarded conclusions is based is as yet far from complete, and I am deeply indebted to Drs Orr and Rows for allowing me to refer to this view: its possible significance in relation to the determining cause of disseminated sclerosis cannot be overestimated.

(To be concluded.)

Abstracts

ANATOMY.

MORPHOLOGICAL INVESTIGATIONS UPON THE CONVOLUTIONAL PATTERN OF RELATIVE BRAINS IN MAN. (302)

F. SANO, *Proc. of Roy. Soc. of Med.*, 1917 (Sect. of Psychiat.), pp. 21-64.

FIFTEEN pairs of adult brains related by direct parentage, and one of twin brains, were examined according to the author's morphological and statistical method.

Eighty-three questions dealing with the convolutional pattern were answered in the case of each pair of brains, and the results summarised and divided according to their kind of similarity and dissimilarity.

A control of comparisons with non-relative brains demonstrated that relative brains are more like one another than non-relative brains, and racial influences (a pair of negro brains being tested) do not play an important part in the results.

Heavy adult brains show less resemblance to the average pattern than the smaller brains. The weight of the brain has no individual value, and it does not influence the essential family resemblance.

The investigations do not permit of the assertion that a given brain belongs to a male or female human being, though comparisons of mixed sexes show less similarity than do comparisons between brains of the same sex.

In the case of the brains of Hebrew brothers and in those of the negroes there were fewer variations in the pattern than in the brains of most of the British subjects, the familial origin of which were in all probability more mixed in type.

There are proofs of differentiation between left and right hemisphere, and the comparison of the hemispheres in the same brain with each other is worthy of careful attention; the amount of variations remaining nearly the same, an increase of differentiated conditions in one part of the brain is an indication of a greater resemblance in other parts.

The author used the same method for each of the questions to be solved regarding the convolutional pattern as he used for the whole brain comparison, and was able to determine which part of the cortex is of more recent development in the ontogeny (which is proved by the comparison of foetal brains), and also in the phylogeny (as suggested by comparative anatomy).

The study of relative brains is of special value in the determination of the transitions from one brain pattern to another both as a whole and in detail. A knowledge of the normal transitions from one convolutional pattern to another may be of use in the consideration of the convolutional pattern in pathological conditions.

The family resemblance of the convolutional pattern in man has an individual value. The family inheritance has a greater effect on the convolutional pattern than is exercised by the influence of brain weight, or by sexual or racial influences.

Considering that there is a constant resemblance shown for the hemispheres in man, we may believe that the apparent differences between the hemispheres of one brain consist of an amount of fixed and inherited differences and an amount of variable conditions, each of which can be revealed by the examination of brains related to the brain in question.

In relative brains the transition from one extreme pattern to another is imperceptible; there is continuity in the evolution.

(This is an exhaustive paper, and should be read in the original. Numerous diagrams and tables are appended, as also a chapter in which the author compares his results with those obtained by other observers.)

H. DE M. ALEXANDER.

AN ANATOMICAL CONSIDERATION OF THE CEREBRO-SPINAL FLUID. LEWIS H. WEED, *Anat. Record*, 1917, xii., May 20, p. 461.

FROM a review of the evidence—histological, pharmacological, and physiological—the writer says the function of the cells of the choroid plexuses as the elaborator of a characteristic body fluid, the cerebro-spinal fluid, seems established. It must be understood, however, that these structures, while undoubtedly producing by far the greatest portion of the cerebro-spinal fluid, constitute merely the intra-ventricular mechanism for fluid-elaboration. There is also further production of cerebro-spinal fluid by the nervous tissue itself—a small addendum poured by way of the perivascular channels into the subarachnoid spaces. Furthermore, a minimal production by the ependymal cells, negligible in its significance and total amount, may occur.

The cerebro-spinal fluid, elaborated by the choroid plexuses, is poured into the cerebral ventricles: that portion which is formed in the lateral ventricles escapes into the third ventricle and thence by the aqueduct into the fourth ventricle. Likewise, an ascending current of fluid apparently occurs in the central canal of the spinal cord; this, representing a possible product of the ependyma, may be added to the intraventricular supply.

From the fourth ventricle the fluid is poured out into the subarachnoid spaces; there is no evidence that functional communications between the cerebral ventricles and the subarachnoid spaces exist in any region except from the rhombic ventricle. As to the exact mode of escape of the fluid from the fourth ventricle into the subarachnoid spaces, the weight of evidence inclines towards the consideration of the foramen of Magendie as a true functioning communication in the inferior velum. The two lateral foramina, those of Luschka, connecting the lateral recesses of the fourth ventricle with the subarachnoid spaces, seem to have as actual an existence as the medial opening of Magendie.

The writer reviews the evidence which favours the existence of a second source of the cerebro-spinal fluid—the extra-ventricular source—from the nervous tissue itself. In 1914, from an analysis of his own experiments on spinal subarachnoid injections of an isotonic solution of foreign salts, Weed concluded that the fluid current in the perivascular system was from nerve-cell to subarachnoid space, and that by this way a small addendum of cerebro-spinal fluid drained into the meningeal spaces. "Such a conception of a dual source of the cerebro-spinal fluid has received support from other observations than those recorded in the foregoing paragraphs. Jacobson's important chemical studies (unpublished) have demonstrated a distinct difference between the subarachnoid fluid (product of choroid plexuses and perivascular system) and the ventricular fluid (product of choroid plexuses alone). The former fluid is richer in protein and poorer in sugar than the latter—a finding to be expected if the products of nerve-metabolism are poured into the subarachnoid space. Likewise, distinct serological differences between subarachnoid and ventricular fluids from the same patient have been reported. And pathologically the occurrence of intra-cortical cysts from dammed-up perivascular channels indicates strongly a production of fluid within the nervous tissue itself, draining outward into the subarachnoid spaces." On the question of the absorption of the cerebro-spinal fluid, Weed concludes, from his review of the evidence, that the absorption of cerebro-spinal fluid is a dual process, being chiefly a rapid drainage into the great dural venous sinuses, and in small part, a slow escape into the true lymphatic vessels, by way of an abundant but indirect peri-neural course. This excellent, learned paper ends with a section on the embryology of the cerebro-spinal spaces, and a full discussion of the subject of the cerebro-spinal fluid. Weed shows clearly that, in the light of modern views as to the nature of lymphatic vessels, there are no lymphatic vessels in the three meninges nor in the nervous tissue itself: true endothelial-lined lymphatic vessels do

not occur in the cerebro-spinal axis. "In the place, possibly, of the true lymphatic vessels, there occur in the nervous tissue the perivascular spaces." And, as no true endothelial-lined lymphatic vessels apparently exist within meninges or central nervous system, the cerebro-spinal fluid cannot be regarded as lymph. Weed refers briefly to the analogies between the processes of the cerebro-spinal fluid and of the aqueous humour of the eye; and mentions that recently Streeter has modified the terminology of the spaces about the ear, terming the so-called perilymphatic spaces the "periotic" (*Amer. Journ. of Anat.*, 1917, xxi., p. 299). The paper by Weed has an excellent bibliography, and is well worthy of close study.

LEONARD J. KIDD.

PHYSIOLOGY.

THE METABOLIC AND VISCERAL CENTRE IN THE INTER- (304) BRAIN: ITS RELATION TO INTERNAL SECRETION (PITUITARY, PINEAL) AND TO DIABETES INSIPIDUS.

(Ueber das Stoffwechsel—und Eingeweide—centrum im Zwischenhirn, seine beziehung zur inneren sekretion (hypophyse, zirbeldrüse) und zum diabetes insipidus.) BERNHARD ASCHNER, *Berliner Klin. Wochschr.*, 1916, liii., No. 28, p. 772 (2 figs.).

Conclusions:—

1. It has not yet been satisfactorily proved that the blood-pressure raising action of pituitrin depends on the pars intermedia and not on the pars nervosa.
2. The same is true of its diuretic action.
3. It is certain that the pars intermedia has nothing to do with fat metabolism, albumen metabolism, respiratory metabolism, inhibition of growth, and genital disturbances; these effects belong especially to the action of the anterior lobe of the pituitary.
4. The indubitable experimental diuretic action of pituitrin (posterior lobe with the addition of an insignificantly small action of the pars intermedia) is related to the interesting observations of Hoppe-Seyler and others, that diabetes insipidus and polyuria are inhibited, and not produced by pituitrin.
5. But there are certain new factors that need consideration, namely, the "vegetative centre of the inter-brain" postulated by Aschner.
6. In favour of the existence of such a vegetative centre there is a series of earlier established facts, namely, a temperature centre in the corpus striatum, Eckhardt's centre for water-regulation in the corpora mamillaria. Secondly, there is the hypothalamus

diabetic-puncture described by Aschner, and also the following effects obtained by mechanical or electrical stimulation of the tuber cinereum, namely, manifestation of severe pain, slowing of pulse up to stoppage of heart, increase of blood pressure, painful respiration, contractions of the pregnant uterus, bladder, intestine, &c. Thirdly, the sympathetic centre in the tuber cinereum (discovered simultaneously and independently by Karplus and Kreidl and by Aschner), stimulation of which in the cat gives dilatation of pupils and secretion of sweat.

7. This inter-brain centre for growth, metabolism, regulation of temperature, and genital development, is concerned not merely with diabetes insipidus, but also with all vegetative disturbances, possibly also with psychical disturbances (migraine) due to cerebral diseases and affections of the pituitary and the pineal bodies.

LEONARD J. KIDD.

A COMPARISON OF THE CHEMICAL COMPOSITION OF THE
(305) **GREY MATTER OF THE HUMAN CORTEX WITH THAT**
OF THE SUB-CORTICAL GANGLIA. (Comparison de la
composition chimique de la substance grise de l'écorce et des
ganglions de l'encéphale chez l'homme.) A. K. LENTZ, *Réun.*
Biol. de Petrograd, 8 Mai 1917; *Compt. rend. Soc. de Biol.*, 1917,
lxxx., p. 753.

FROM a study of ten human brains the writer reaches these conclusions:—

1. The grey matter of the cerebral cortex differs from that of the sub-cortical ganglia (corpus striatum was specially studied), by containing a larger amount of water, total nitrogen, and albuminoid substances, especially neuroglobuline, and a smaller amount of lipoids.

2. The cortex and the basal ganglia resemble each other in the structure of their albuminoid portion. One must regard the cortex as composed of the same elements as the ganglia, but richer in water and poorer in lipoids.

3. The predominance of albuminoid substances in the cortex, as compared with the ganglia, and their diminution from the centre towards the periphery, shows the importance of the part played by these albuminoids in brain work. The lipoids play a secondary part.

The writer recommends the method used by him in the study of albuminoid substances and their modifications in the brain, viz., that of extraction of the neuroglobuline by weak organic acids.

LEONARD J. KIDD.

THE INFLUENCE OF THE VAGUS NERVE ON INTESTINAL
(306) **SECRETION.** (L'influence du nerf vague sur la sécrétion de l'intestin.) V. V. SAVITCH and N. A. SOCHESTVENSKY, *Réunion Biol. de Petrograd*, 17 Janvier 1917; *Compt. rend. Soc. de Biol.*, 1917, lxxx., 19 Mai, p. 508.

THE writers describe the technique of their experiments on cats anæsthetised by ether or chloroform. Electrical stimulation of the vagus in the neck gives very quickly a peristalsis of the intestine which diminishes in two hours and then ceases. A considerable secretion of intestinal juice then begins. Intravenous injection of 10 mgm. of atropine interrupts, or at any rate lessens, this secretion: but subsequent vagus stimulation causes a fresh secretion. The writers conclude that the vagi nerves contain secretory fibres which regulate the secretion of the intestine. But, as rather a long period of electrical stimulation of the vagi is necessary to provoke intestinal secretion, they believe that the vagi contain also inhibitory fibres for intestinal secretion.

LEONARD J. KIDD.

EXPERIMENTAL OPERATIONS ON THE PITUITARY. W. BLAIR
(307) BELL, *Quart. Journ. Exper. Physiol.*, 1917, xi., pp. 77-126 (57 figs.).

THESE experiments were performed on twenty-five female dogs, most of which were from four to seven months old, while a few were a little older. Conclusions:—

1. The pituitary body is essential to life: its removal causes death within a few hours. In the cases which survive for longer periods the removal has probably not been complete.

2. The removal of very large portions of the pars anterior is incompatible with life. It appears certain from the evidence at our disposal that it is the loss of this portion of the organ which proves fatal when total extirpation of the pituitary is practised.

3. Partial removal of the pars anterior may, if sufficient quantity be removed, cause genital atrophy. This may occur in the absence of any other symptom, although the animal may also remain undersized.

4. Neither partial nor complete removal of the pars posterior causes any symptom. The genital organs remain normal after operation, and young animals continue to develop. Hence the secretion of the pars nervosa is neither necessarily beneficial nor essential to life.

5. Partial removal of the partes anterior and posterior causes no symptom, provided only a small portion of the pars anterior be removed.

6. Clamping and separation of the infundibular stalk, by interfering with the blood supply, and so causing degeneration in the

cells of the partes anterior and intermedia, lead to the condition known as dystrophia adiposo-genitalis.

7. Artificial tumours in the neighbourhood of the sella turcica may produce irritation, which is accompanied by glycosuria and emaciation; or by interfering with the blood supply may lead to degenerative changes in the cells of the pars anterior, and so give rise to the syndrome dystrophia adiposo-genitalis.

8. The pituitary body appears to be one organ and not two; and the essential and beneficial secretion is taken up by the blood stream, as in the case of the other organs of internal secretion.

LEONARD J. KIDD.

EFFECTS OF THE EXTIRPATION OF THE ANTERIOR LOBE (308) OF THE HYPOPHYSIS IN *RANA PIPIENS*. BENNET M.

ALLEN, *Biological Bull.*, 1917, **xxii.**, March, p. 117 (4 figs.).

A RECORD of a large number of experiments on tadpoles of *Rana pipiens*. Conclusions:—

1. The removal of the anlage of the anterior lobe of the hypophysis early causes the pigment cells to contract, and those of the epidermis to withdraw from it into the interior. This takes place while the gland shows little or no apparent histological differentiation.

2. Evidence has been given to show that the absence of the hypophysis in tadpoles makes them highly susceptible to unfavourable chemical conditions of the water. This will be tested out specifically in later experiments.

3. The absence of the hypophysis causes—either directly or indirectly—a failure to undergo metamorphosis, especially evident in the absence of limb growth beyond a very rudimentary condition.

4. The absence of the hypophysis causes a marked diminution of colloid formation in the thyroid gland, and in the stage of 24 mm. involves a retardation in its growth.

5. During the stages studied, the absence of the hypophysis produced no noticeable effects upon the thymus gland nor upon the gonads.

In nearly every experiment where removal of the hypophysis was performed one or two operated tadpoles failed to show the colour change characteristic of the others. Several of these were sectioned, and in each case it was found that there had been failure to remove the hypophysis. "There is thus no doubt whatever that the absence of the gland is the cause of it." A few days after the operation the hypophysectomised tadpoles changed from a solid black to a bright creamy silver colour.

LEONARD J. KIDD.

THE ACTION OF ADRENALIN ON THE DIGESTIVE TRACT.

(309) (*L'action de l'adrenaline sur le tractus digestif.*) M. LOEPER and G. VERPY, *Compt. rend. Soc. de Biol.*, 1917, lxxx., p. 703.

THE gastric activity and the digestive motility were determined in eight subjects: an intra-muscular injection of 1 mgm. of adrenalin was then given. Test-meals were taken half an hour, one hour, and an hour and a half after the injection. The following results were obtained:—

1. Adrenalin increases the total hydrochloric coefficient, and especially the amount of free HCl in the stomach: this augmentation, barely perceptible for the meal absorbed in the half hour, reached its maximum in the hour, and diminished in the hour and a half which followed the injection.

2. Adrenalin modifies the gastric contractility: radioscopy shows that in hypotonic subjects there is increase of the frequency and intensity of the contractions, whereas in hypertonic subjects the contractions are regularised and the spasms and constrictions are removed.

3. Adrenalin accelerates the passage of the contents of the digestive tract.

The writers point out that this secretory and motor action of adrenalin on the gastro-intestinal tract enables us to suspect that there are certain digestive syndromes which are of adrenal origin and need adrenal therapy. (Loeper and others have recently described an adrenal dyspepsia.)

LEONARD J. KIDD.

EXPERIMENTS WITH FEEDING THYMUS GLANDS TO FROG

(310) **LARVÆ.** W. W. SWINGLE, *Biol. Bull.*, 1917, xxxiii., Aug., p. 116.

GUDERNATSCH found in 1912, and again in 1914, that fresh thymus gland, when fed to tadpoles, stimulates their growth processes, and at the same time inhibits the onset of metamorphosis. Most of the experimentalists who have performed thymectomy in various young mammals have failed to find any changes in their growth. Adler extirpated the thymus of tadpoles, and found no subsequent ill effects, and no modification of their growth and development. Swingle has now found that feeding thymus gland, either in the fresh state or in powder, to larvæ of the species *Rana pipiens*, *R. catesbiana*, and one undetermined species, does not accelerate the growth processes, or retard their metamorphosis. The tadpoles seem to develop normally in every respect. The gonads do not appear to be affected by thymus feeding. His results thus disagree with those of Gudernatsch: he thinks that possibly the species of frog used by that observer may react differently to thymus feeding from those used by himself. But

Swingle finds that even very slight environmental changes produce fluctuations of growth in frog larvæ; he thinks, therefore, that Gubernatsch's results may be, in part at least, due to some other factor than thymus feeding. Also, in thymus feeding experiments the growth-accelerating principles of the thymus, if there be such, may possibly undergo disintegration, or other change, by contact with digestive enzymes in the alimentary canal. At the same time, no such changes occur in thyroid feeding experiments.

LEONARD J. KIDD.

PSYCHOLOGY.

THE SYMBOLISM OF CERTAIN DREAMS OCCURRING DURING (311) PULMONARY TUBERCULOSIS. (A fragment of auto-psych-analysis.) CH. BAUDOUIN, *Archiv. de Psychol.*, Dec. 1916, p. 133.

A DESCRIPTION of eight dreams, with a commentary on each showing the "latent content." Two consequences seem to the author to follow from his analysis: first, that the dreams were determined by his psychic, not by his physical state; and second, that the psychologists who tend to abandon the "pansexual dogma" of Freud are right, for the "repression" indicated by these dreams has nothing to do with the *libido*, but merely with the *will to live*.

MARGARET DRUMMOND.

PSYCHANALYSIS OF SOME NERVOUS SYMPTOMS. CH. BAUDOUIN, (312) *Archiv. de Psychol.*, Dec. 1916, p. 143.

THIS is a description of two cases treated by psychanalysis. In the first case the symptoms were violent pains in head and stomach, neuralgia of the left arm, and the delusion of persecution. Great improvement was effected by suggestion, supplemented by auto-suggestion; the remaining symptoms were resolved by psychanalysis. In the second case there were periodic disturbances sometimes marked by phobias, hallucinations, and incoherent speech. Here also the writer succeeded in tracing the trouble to its source; and a cure was effected chiefly by a "sublimation" of the harmful activities in the direction of art.

MARGARET DRUMMOND.

THE STRUCTURE OF THE UNCONSCIOUS. C. G. JUNG, *Archiv. de Psychol.*, Dec. 1916, p. 152.

THOSE who are interested in the theory of psychanalysis will find much to interest them in this paper of Dr Jung's.

It is well known that Dr Jung does not subscribe to Freud's *pansexual dogma*. He considers that the mental disturbances

propagated from the unconscious demand for their explanation a searching examination of the nature of the unconscious.

According to Freud, the unconscious is made up of personal elements which have been repressed because they are incompatible with the main trend of the character. Jung points out that this is a very imperfect concept of the unconscious, which must also contain psychic elements which have not yet attained the threshold of consciousness, together with others which never will pass the threshold of consciousness, subliminal sense perceptions, for example.

These elements, like the ones distinguished by Freud, are personal; but penetration to a deeper stratum of the unconscious reveals impersonal or collective elements. These collective elements comprise the fundamental modes of thought, feeling, and activity common to all men. It is to their presence that we must attribute the similarity of the myths and symbols of all races and all countries.

When this region of the unconscious is penetrated by psychoanalysis certain morbid phenomena, as delusions of greatness or the contrasting sentiment of incompetence, are liable to appear. It now becomes the function of the analyst to re-establish the balance between the conscious and the unconscious by directing the patient's energy along certain "lines of life" revealed by the study of the personality. This is not possible unless the patient himself takes an active part in his own treatment. Dr Jung protests against the idea that "analysis" alone can bring about cure.

MARGARET DRUMMOND.

PATHOLOGY.

CLINICAL AND PATHOLOGICAL CONTRIBUTION TO THE

(314) **QUESTION OF MOTOR LOCALISATION IN THE SPINAL CORD.** (*Contributo clinico ed anatomopatologico alla questione delle localizzazioni motrici spinali.*) E. TROCELLO, *Riv. di patol. nerv. e ment.*, 1917, xxii., p. 389.

A RECORD of a case of a man in whom disarticulation of the thumb at the metacarpo-phalangeal joint had been performed at the age of 21. Death from general paralysis took place at the age of 39. Careful histological examination of the cervical enlargement and first dorsal segment showed no hemiatrophy of the white or grey substance, or any difference in staining on the two sides. The various cell groups were equal in number and size on the two sides, the nerve fibres were well preserved, and the neuroglia and vessels showed no change.

J. D. ROLLESTON.

- DETAILED STUDY OF A MONSTER WITH CRANIO-RACHI-**
 (315) **SCHISIS AND OTHER ANOMALIES.** CARBON GILLASPIE and
 HOWARD HULL HEUSTON, *Anat. Record*, 1917, xiii., Oct., p. 289.

THE subject of this report was a foetal monster of some eight months. It presented quite the usual appearance of the anencephalic monsters. A detailed study of its anatomy revealed the presence of numerous anomalies. The nerve-supply of the extremities was apparently normal, also the brachial and the lumbo-sacral plexuses. The arms and hands were abnormally large. The condition of cranio-rachischisis is figured. There was no brain and no spinal cord. The bones of the cranial cavity and of the spinal canal were covered only by the membrane which formed the floor of the embryonic neural groove. "It is interesting to note that while neither brain nor cord was formed, there was a complete and apparently normal development of the peripheral nervous system." (The condition of the vegetative nervous system is not mentioned.) LEONARD J. KIDD.

- THE INFLUENCE OF CEREBRAL EMBOLISMS ON BLOOD PRES-**
 (316) **SURE.** (*Influence des embolies cérébrales sur la pression sanguine.*) H. ROGER, *Compt. rend. Soc. de Biol.*, 1917, lxxx., 21 Avril, p. 377.

ROGER produced cerebral embolisms in rabbits and dogs by injecting lycopodium into the common carotid artery, with the result that a very great rise of arterial tension was almost constantly produced after a slight temporary fall. When these injections are repeated the tension is still further increased; but if this be done very often the final result is a secondary fall of arterial tension.

LEONARD J. KIDD.

- THE RÔLE OF THE ADRENALS IN THE ARTERIAL HYPER-**
 (317) **TENSION WHICH FOLLOWS CEREBRAL EMBOLISMS.**
 (*Le rôle des surrénales dans l'hypertension artérielle consécutive aux embolies cérébrales.*) H. ROGER, *Compt. rend. Soc. de Biol.*, 1917, lxxx., 5 Mai, p. 427.

THE arterial hypertension which follows the production of experimental cerebral embolisms in rabbits and dogs lasts for many hours, but is not permanent. Roger's further experiments were performed to determine whether this hypertension is due to excess of adrenalin secretion. He extirpated the adrenals in rabbits by the abdominal route. On injecting lycopodium into the peripheral end of the common carotid artery he found, as in normal animals, first a temporary slight fall of arterial tension; then the animals became agitated, and a marked rise of tension

followed. But it gives place almost immediately to a fall below the initial level.

From these two sets of experiments Roger finds that cerebral embolism, experimentally produced, excites convulsive movements which cause a considerable rise of arterial pressure. But as this rise is not maintained in the adrenalectomised animals, he concludes that the hypertension in the normal animals is due to a secondary action of the adrenals, in the shape of an excess of adrenalin induced by the cerebral embolism.

LEONARD J. KIDD.

GENERAL.

AN IMPROVISED REFLEX HAMMER. (*Marteau à réflexes improvisé.*) PERRIN, *Paris Méd.*, 1917, vii., p. 81.

THIS can be constructed with a large piece of indiarubber weighing 25-30 gm. (price 50 centimes) and a metallic stem such as a knitting or crochet needle about 20 cm. long (price 25 centimes).

A hole must first be bored in the indiarubber with a thermocautery, bodkin, or nail, and the stem inserted. If necessary, a cork can be fixed at the other end to serve as a handle.

J. D. ROLLESTON.

THE OCULO-CARDIAC REFLEX AND EXTRA-SYSTOLES.
(319) **DAGNINI'S SIGN.** (*Riflesso oculo-cardiaco ed extrasistoli. Fenomeno del Dagnini.*) G. FERRALIS and C. PEZZI, *Le Malattie del cuore e dei vasi*, 1916, i., p. 36.

THE priority for this sign should be assigned to Dagnini, who first described it at the Medico-Chirurgical Society of Bologna on 17th June 1908, whereas Aschner, who is usually credited with its discovery, published his paper on 29th October 1908.

The writers, as the result of numerous experiments with the electrocardiograph, have found that bilateral ocular compression produced extra-systoles either isolated or in series.

Until a more precise explanation can be offered, they explain this occurrence by saying that the vagus may create a special condition of the myocardium which facilitates the appearance of premature contractions.

J. D. ROLLESTON.

ACUTE ASCENDING TOXIC (OR DEGENERATIVE) MYELITIS.
(320) **Report of a case with necropsy findings.** JULIUS R. GLOBUS, *Journ. Amer. Med. Assoc.*, 1917, lxi., Sept. 8, p. 816.

A MAN, aged 31, of good antecedents, was admitted to hospital on 3rd May 1917. He had come home the previous evening

slightly under the influence of alcohol, but otherwise well. On the morning of 3rd May there was a complete flaccid palsy, with the exception of the right fingers and right shoulder; all trunk muscles paralysed, and breathing purely diaphragmatic. Neck slightly rigid, but he could move head freely in all directions, but with slight pain; otherwise there was no pain whatever. Bladder greatly distended; constipation; all reflexes lost. Sensation completely lost in both lower extremities and trunk, and markedly diminished in both upper limbs; only slightly, if at all, in lower neck. Temperature and pulse both rose, and respiration to less degree. Patient was perfectly conscious throughout. An hour before death he began to lose control over the muscles of articulation and deglutition. In from 36 to 42 hours from the appearance of his first symptoms he suddenly became deeply cyanosed, and died, after a few minutes, from respiratory paralysis. Necropsy showed a very soft and spongy condition of bulb and cervical and upper dorsal cord; the anterior part of the cord was congested. Brain normal. Spleen somewhat large and congested; other organs normal. Histological examination showed a normal general topography of the cord; spinal meninges normal, except for slight vaso-dilatation. Vacuolation of ventral horn cells, which in places had gone on to cell-fragmentation. Generalised œdema of ventral horns, extending to dorsal horns; blood vessels markedly dilated, but no round cell infiltration. Outside the grey matter there was a general œdema of the nerve-fibres and neuroglia, without round cell infiltration. The bulb showed a generalised œdema, with absence of hæmorrhage, round cell infiltration, or other evidences of inflammation.

LEONARD J. KIDD.

OTHER FORMS OF SPEECH IN TABES. WALTER B. SWIFT, *Proc. of* (321) *Amer. Med.-Psychol. Assoc.*, 1916, April 4-6.

Two cases of tabes show the speech sign in form differing from that reported last year. One case shows slowness and frequent elision of sounds that require exact muscle co-ordination; the other shows a pervasive slovenliness. The previous report (1) of the sign is confirmed; and the method reported (2) for a technique of elicitation found applicable.

AUTHOR'S ABSTRACT.

THE RELATION OF MOSQUITOES AND FLIES TO THE EPI-
(322) **DEMOLOGY OF ACUTE POLIOMYELITIS.** HIDEYO NOGUCHI
and ROKUSABURO KUDO, *Journ. of Exp. Med.*, 1917, xxvi., July,
p. 49.

SUMMARY of the experimental findings of the writers:—

(1) *Culex pipiens* raised from the larval stage in water experi-

mentally contaminated with an abundance of poliomyelitic virus were found to be incapable of causing the infection when allowed in large numbers to bite normal *Macacus* monkeys.

(2) *Culex pipiens*, which were fed on infected poliomyelitic monkeys during different stages of the disease, were found to be incapable of transmitting the infection when allowed in large numbers to bite normal *Macacus* monkeys. A previous disturbance of the meninges by an injection of horse serum into the intrathecal space did not alter the result, which was negative.

(3) The offspring of the mosquitoes, which were either reared in the infected tanks or fed on infected monkeys, were found to be entirely harmless when allowed to feed in large numbers on a normal monkey. There was no hereditary transmission of the virus from one generation to another.

(4) No trace of the virus of poliomyelitis was demonstrable in the filtrate of an emulsion of adult flies and pupæ of the common house fly and bluebottle fly which were reared in the laboratory on slices, emulsion, or filtrate of monkey brain containing the poliomyelitic virus. The intra-cerebral injection of the filtrate produced no poliomyelitic infection in the normal monkey.

LEONARD J. KIDD.

MECHANISMS THAT DEFEND THE BODY FROM POLIO-
(323) **MYELITIC INFECTION, (a) EXTERNAL OR EXTRA-**
NERVOUS, (b) INTERNAL OR NERVOUS. SIMON FLEXNER,
Proc. National Acad. of Sci., U.S.A., 1917, iii., June, p. 416.

"IN view of the wide distribution of the virus of poliomyelitis and the relatively low case incidence, we must suppose that many more persons are exposed to, than acquire, the infection. Hence the body must possess defensive mechanisms usually sufficing to protect it from invasion. Two sets of defences have been detected:—

"(1) The first or external consists of the nasal and probably pharyngeal mucous membranes. The secretions in many, if not in most persons, when left in contact for a relatively short time with the virus, inactivate or neutralise it. Some persons fail to yield this neutralising nasal secretion; in others a temporary pathological state of the mucous membranes removes the inactivating property previously present. The number of tests is still too small to determine whether young persons, who are the more susceptible, yield secretions which are numerically inferior in neutralising power to those supplied by older persons. Probably the failure of this external defensive measure is not in itself decisive, because of the existence of the second or inner defensive mechanism.

"(2) The second or inner mechanism consists of the membranes about the brain and spinal cord and the attached secreting organ of the choroid plexus. This meningeal-choroid complex is remarkably efficient in excluding from the cerebro-spinal fluid, and hence from the substance of brain and spinal cord, almost everything present in the circulating blood, except water and a few inorganic salts. Only when the complex has been injured in some way and its integrity impaired does it permit even protein and cells to pass through from the blood into the cerebro-spinal fluid.

"Possibly poliomyelitis arises during the prevalence of the malady when both sets of mechanisms fail. This probably would occur only in exceptional instances in individuals among populations of any size. It is for the moment not difficult to conceive of reasons to account for the failure of the external mechanism, and more difficult to account for failure of the internal mechanism of defence. Not improbably the neutralising power of the nasal secretions tends to reduce the carriage of the virus upon the nasal mucosa of persons exposed to and having suffered from infection with the virus of poliomyelitis. It becomes, therefore, an essential agency in diminishing public danger through reduction in the number of the potential virus carriers which arise.

"There is one irritating fluid only so far detected which does not promote infection when injected into the cerebro-spinal meninges. This fluid is an immune serum obtained from monkeys or human beings previously recovered from poliomyelitis. The immune serum carries neutralising principles which inactivate the virus as it passes from the blood into the cerebro-spinal fluid. This observation is in harmony with the curative action exercised by the serum, as was first shown some years ago in inoculated monkeys, and has recently been confirmed for human cases of epidemic poliomyelitis."

LEONARD J. KIDD.

OBSERVATIONS ON THE PRINCIPLES GOVERNING THE
 (324) **EARLY TREATMENT OF INFANTILE PARALYSIS.** W.
 COLIN MACKENZIE, *Brit. Med. Journ.*, 1917, i., Feb. 24, p. 249.

THE question of treatment is here considered from the point of view of (a) rest, and (b) muscular function.

(a) *Rest*.—Anatomical rest of a joint means that the muscles acting on that joint are passively controlled, *i.e.*, the joint or joints with which it is in relation are rendered immobile. A tuberculous ankle is not at rest if the knee be mobile, nor is the knee unless the hip and ankle are fixed. In the upper limb adequate fixation of one joint is best secured by fixation of all three. Details are given for obtaining anatomical rest to the upper or lower limbs.

(b) *Muscular Function*.—The deltoid and quadriceps extensor of the knee are here dealt with. The former is generally considered the most difficult for recovery, while loss of function in the latter is responsible for the swinging limb and crutchdom of any large city. Some interesting points in the evolution of muscular action in the mammalia are described.

A. NINIAN BRUCE.

SENSORY DISTURBANCES IN DIPHTHERIA: DIPHTHERITIC

(325) **HYSTERIA**. (*Troubles de la sensibilité au cours de la diphtérie: l'hystérie diphtérique.*) F. RAMOND and B. DE LA GRANDIÈRE, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 1506.

DURING the last two years the writers have noted the frequency of sensory troubles in their diphtheria patients, both in severe and moderate attacks.

The influence of nervous antecedents appeared to be nil, as they were present in all classes of society. The onset was very early, from the second to the eighth day of the angina. Taste, smell, vision, and hearing were affected in severe attacks, the sensory troubles were early and extensive, while in the mild attacks they were late and were less pronounced. The sensory trouble assumed the form of hemianæsthesia to touch, pain, heat, and cold.

Their duration varied, in some cases they all disappeared in eight to ten days; more frequently they persisted three weeks, a month, or even three months. As a rule the anæsthesia affected only a few areas, always on the same side. Ageusia was the most constant change, as well as various ocular disturbances, then came the disorders of other special senses, and finally those of skin sensation.

The writers hold that their observations favour the real existence of toxic hysteria.

J. D. ROLLESTON.

MENINGEAL HÆMORRHAGES AND OTHER HÆMORRHAGIC

(326) **MANIFESTATIONS IN RECURRENT FEVER**. (*Hémorragies méningées et autres manifestations méningées dans la fièvre récurrente.*) V. BABES, *Compt. rend. Soc. de Biol.*, 1916, lxxix., p. 855.

MENINGEAL symptoms are not rare in recurrent fever, especially in children. Babes records a fatal case in a man, aged 50, who died in a comatose state on the third day of disease. The autopsy showed the presence of meningeal hæmorrhages.

J. D. ROLLESTON.

ACUTE MENINGITIS IN THE NEWLY-BORN. (*Méningite aiguë* (327) *chez le nouveau-né.*) Mdlle. CONDAT, *Arch. de Méd. des Enfants*, 1917, xx., Août, p. 404.

VERY few cases of acute meningitis in the newly-born have been described; in many of these its existence has been found only on necropsy. The authoress claims that it is not really rare, but is commonly overlooked, as its symptomatology is usually incomplete, and the diagnosis is difficult. Digestive and pulmonary signs may be prominent. Its signs are, in fact, often discrete or absent, and it is masked by the general ensemble of the infection. Fever may be the first sign: but temperature may be normal, or even sub-normal exceptionally: hypothermia was very marked in Herrman's case. The authoress gives particulars of three personal cases, one of which was streptococcal, the two others pneumococcal; and also of eight cases collected by Herrman (one being his own case of pneumococcal meningitis). She regards meningitis acuta neonatorum as a frequent secondary complication of infection, and as due in general to the streptococcus, more rarely to the pneumococcus. LEONARD J. KIDD.

OCCIPITAL MENINGO-ENCEPHALOCELE (ECTOPIA OF CEREBELLUM) IN A GIRL AGED TWO AND A HALF MONTHS. (*Méningo-encéphalocèle de la région occipitale (ectopie du cervelet) chez une petite fille de deux mois et demi.*) E. KIRMISSON and TRÉTIAKOFF, *Arch. de Méd. des Enfants*, 1917, xx., Août, p. 412 (3 figs.).

A RATHER puny, bottle-fed infant presented what seemed to be an enormous meningo-encephalocele of the occipital region, situated slightly to right of the middle line. Patient's father unknown: mother has good health, but has a dermoid cyst of the eyebrow. The tumour was biloculated: greatest length 6 cm.; greatest circumference 13 cm. The skin over its free portion was very thin. Its tension was not very great: still, pressure over it was transmitted to the anterior fontanelle. It appeared to be perfectly transparent, and was thought to contain no nerve elements. It was irreducible, and pressure on it failed to evoke convulsions. There were also some palatal anomalies. As rupture was feared on account of the thinness of its coverings, and there was an absence of cerebral symptoms, operation was performed. For ten days things went well: then a severe gastro-enteritis came on, and death took place on the nineteenth day after operation. Necropsy showed that almost the whole of the cerebellum was included in the tumour, so that it was really an ectopia cerebelli. Histological examination proved that the portion of nervous tissue which formed part of the hernia belonged

to the cerebellum. Cerebellar convolutions were found, cut in various directions. On closer examination, two sets of convolutions were found: some corresponded exactly in the structure of their cerebellar layers to those of a full-term child, with their layer of superficial granules, molecular layer, layer of Purkinje cells well developed and provided with axons, the layer of deep granules, and finally a layer of myelinated fibres; but in other places the layer of granules becomes thinned and disappears, and the deep layer of granules is less numerous than normal, Purkinje cells are too small and have hardly their proper shape. A full description of the cerebellar peduncles, pons, and other parts of the brain is given.

LEONARD J. KIDD.

**HEMIPLEGIA AND APHASIA CAUSED BY SUBDURAL
(329) HÆMORRHAGE; OSTEOPLASTIC FLAP; RECOVERY.**

ANTHONY H. HARRIGAN, *New York Acad. Med.* (Sect. Surgery),
Oct. 5, 1917; *Med. Record*, 1917, xcii., Oct. 27, p. 741.

DR HARRIGAN presented the patient, a woman of 40, who was admitted to hospital on 2nd August 1913. He said the injury resulted from an automobile accident, and that, on admission, patient was unconscious, with incontinence of urine. Physical examination negative except for abrasions on face, head, and arms. Slight bleeding from nose, but no depression of vault of skull, Temperature 100, pulse 60 to 30, blood pressure 215. Dr Cornwall diagnosed fractured base. There were, however, no focal symptoms, though there was bilateral Babinski, and knee jerk slightly plus. Next day patient could be slightly aroused. Blood pressure at 11 A.M. was 240, and at 9 P.M. 190 mm. On the next night a hemiplegia developed, with paralysis of right face. Patient could be aroused: asked to shake hands, gave the left readily: could move left leg, but not right. There was a subconjunctival hæmorrhage "out of one-half the right eye." Dr Cornwall advised immediate operation. The Rolandic fissure was outlined on the left side, following the Kronlein method of cranial topography. The flap was outlined over this area. The bone was divided with the Hartley-Kenyon electric saw. On turning the flap down it was found that the dura did not pulsate. About four ounces of black clotted blood was found lying on the motor area; this was removed, and considerable blood-clot was taken from the base. This clot was mixed with disintegrated brain tissue. The finger could detect easily a marked laceration of the brain on the inferior surface of the frontal lobe. The dura was sutured, and the skin closed with interrupted silkworm gut. The patient was in excellent condition, and regained consciousness during the day.

Next day she could move right arm and leg slightly. She continued to improve, though the incontinence of urine persisted for several days; and finally she made a perfect recovery.

LEONARD J. KIDD.

NOTES ON A CASE OF CYST IN THE THIRD VENTRICLE.

(330) D. MAXWELL ROSS, *Journ. Ment. Sci.*, 1917, April, p. 252.

A WOMAN, 55 years of age, exhibited for about two years symptoms suggesting an organic intracranial lesion. She died suddenly. The post-mortem examination revealed a simple retention cyst situated in the anterior part of the third ventricle with softening of the centre of the body of the corpus callosum, atrophy of the body and adjacent anterior pillars of the fornix, and unusually free communication between the third and lateral ventricles. During life the physical and mental symptoms corresponded reasonably closely with those exhibited in the cases of tumour of the third ventricle collected by Weisenburg; but the latter asserted the mental symptoms arose from increased intracranial pressure. The mental symptoms in this case were probably due to the direct destructive pressure of the cyst on the surrounding tissues.

H. DE M. ALEXANDER.

AN OBSCURE CASE OF SUB-DURAL ABSCESS OF THE BASILAR

(331) **FRONTAL AND LEFT TEMPORAL AREAS.** OTTO G.

FREYERMUTH, *Pacific Med. Journ.*, 1917, ix., Oct., p. 538.

PATIENT was a single man of about 45: admitted in semi-conscious state. No data obtainable of past history, present illness, or family history. The tentative diagnosis on admission was a possible meningitis. No scars or traces of injury: much emaciated: skin sallow and dry: lax dorsal decubitus: no restlessness or apparent distress: expression apathetic: cannot speak, write, or express himself by countenance. Viscera normal, also temperature and pulse: blood pressure 135 and 80: slight arterio-sclerosis, perhaps not more than physiological. Sensibility not testable. All reflexes normal. Right optic disc shows a suspicion of congestion: left disc, a pronounced central area of atrophy—a secondary atrophy. Muscles flaccid. Apathy and lethargy. Mental state ranges from semi-consciousness to stupor. Blood and spinal fluid negative Wassermann and Noguchi: no abnormal spinal fluid pressure. Cell count 12, the globulin negative. Urine high coloured, but not pathological. Diagnosis uncertain: possibly intra-cranial neoplasm. Death in a week. *Necropsy*:—Dura much congested: on opening it, much pus exuded from left side. The whole left cerebrum bathed in pus. Cortex much congested,

arterioles pronounced. Abscess cavity in base of left frontal lobe, a small channel extending backwards to the base of the temporal lobe and the contiguous upper border of left cerebellar lobe, the tentorium cerebelli forming the septum. Right brain normal: no intra-cerebral or ventricular changes. During life an exploratory decompression was seriously considered, but diagnosis was very difficult, and nothing pointed definitely to abscess. The writer thinks that whenever confused mentality is accompanied by choked discs, whether in traumatic or non-traumatic cases, decompression should be performed. Possibly relief might have followed an immediate decompression in this case, but the writer feels that, on account of the extensive cerebral involvement present, no benefit would have been gained.

LEONARD J. KIDD.

THE DIFFERENTIAL DIAGNOSIS BETWEEN OTOGENIC (332) TEMPORAL LOBE ABSCESS AND PITUITARY TUMOUR.

(Zur differenzial diagnose zwischen otogenem Schläfelappen-abscess und hypophysentumor.) G. ALEXANDER, *Wien. Klin. Wochschr.*, 1916, xxix., p. 767 (1 fig.).

THE writer records a case of pituitary tumour which, in almost all of its symptoms, pointed to otogenic temporo-sphenoidal abscess. A soldier, aged 19, had had middle-ear disease since childhood. For a few weeks before admission he had had headaches and increase of aural discharge. On admission, right ear full of foetid pus, tympanic membrane almost entirely destroyed, copious flow of pus from antrum and attic, hearing power 0 on right, no signs of labyrinthine irritation, lessening of bone conduction, and diminution in perception of high tones on right. Temperature normal, pulse 58. Headaches, vomitings, sensorium clouded at times, moderate stiffness of neck. On the third day fundus oculi normal. Complete peripheral paralysis of right oculomotorius, with escape of second, fourth, and sixth nerves. On the fourth day radical operation. A focus of pachymeningitis, the size of a bean, on the outer surface of the dura over the tegmen antri. Incision of dura. Immediate prolapse of temporal lobe (size of walnut). Incision into the oedematous brain substance gave vent to no pus. No flow of cerebro-spinal fluid from the endodural cavity. Death six days after operation. *Necropsy*:—An adenoma of the pituitary, growing forward into the cavernous sinus. The right oculomotor nerve passed into the tumour like a peduncle. Sub-dural and third ventricle—bleeding; cerebral oedema. In his discussion the writer points out that in this case there were no signs of acromegaly, of adiposo-genital degeneration, or of visual symptoms, which might point to pituitary tumour. The lack of visual symptoms is explained by the fact that the tumour had grown backwards and

laterally towards the middle cranial fossa. The only symptoms that favoured diagnosis of pituitary tumour were the somnolence, the psychical listlessness, and the relatively low temperature. The oculomotor palsy might favour either temporo-sphenoidal abscess or pituitary tumour.

LEONARD J. KIDD.

THE INFUNDIBULAR SYNDROME IN A CASE OF TUMOUR (333) OF THE THIRD VENTRICLE. (*Le syndrome infundibulaire dans un cas de tumeur du troisieme ventricule.*) HENRI CLAUDE and J. LHERMITTE, *Presse Méd.*, 1917, Ann. xxv., No. 41, 23 Juillet, p. 417 (2 figs.).

THE patient was a man of 25, who had a chancre at 20, and was treated for four months in hospital in April 1916 for anaemia: he came under the writers' care in September 1916; he was then ill, wasted, and pale; he had had anorexia for many months and had become feeble. The asthenia was pronounced: he slept badly and had polydipsia, polyuria, but no polyphagia nor glycosuria. The spleen seemed slightly enlarged. There was progressive amblyopia so that reading was difficult, and he could tell only the larger letters. Lumbar puncture showed a clear fluid under slight hypertension, containing 0.56 of albumen and numerous lymphocytes. There was typical complete bitemporal hemianopia, but no papilloedema nor ocular palsies. The nasal half of each iris was pale, especially on the left side: both pupillary reflexes diminished: on the left side barely perceptible. The pupils showed great variability in size, being sometimes very large, at others very small. Patient was put on intensive specific treatment: a few days later his pulse became arrhythmic, and there were extrasystoles at times. Eight days after this treatment was begun, speech troubles appeared: it was slow, scanning, drawling, and monotonous. Anti-syphilitic treatment was stopped, and the articulatory troubles soon went. On 23rd October patient fell into a profound sleep from which he could not be roused. This narcolepsy lasted about five hours, and left him amnesic and astonished. There was a general absence of other nervous symptoms, with the exception of disturbances of memory which lasted only a short time and disappeared a few days after the narcoleptic crisis. On 26th November cardio-vascular symptoms reappeared, with a pulse of 136 of embryonic type, and feebleness of heart-beats. On 30th November blindness. The polyuria persisted, from 2½ to 3 litres per twenty-four hours. On 26th December great cachexia, with definite signs of pulmonary tuberculosis at right apex, and a confusional delirium with a dreamy state ("onirisme"). There was disorientation in time and space, and euphoria. No marked changes occurred up to his death

on 17th February 1917. Necropsy showed a fluctuating retro-chiasmatic swelling of violet colour. The pituitary was normal, also the sella, and it was not apparently compressed. The inner part of the optic tracts was manifestly flattened. Histological examination revealed a cystic epithelial tumour in connection with the third ventricle: that ventricle was distended by the tumour which had thinned out especially the inferior segment of the ventricle, the infundibulum, and the lamina terminalis, but had spared the pituitary entirely. The lateral ventricles were slightly distended. No meningeal nor vascular changes were found.

The writers have an interesting discussion of their importance with reference to recent experimental researches, especially those of Aschner and of Camus and Roussy. They point out that in man, as in dogs, lesions of the ventral part of the third ventricle may be accompanied by profound disturbances of the circulation and of the regulating mechanism of the hydration of the tissues. In the appearance of these phenomena the pituitary body has but an indirect rôle. As to the nature of these centres in the infundibulum or the tuber cinereum, only hypotheses can at present be offered. But the facts of the writers' case and of the experimentalists just named speak in favour of the theory of the modern anatomists, viz., of a series of centres, arranged at intervals from the bulb up to the infundibulum, whose action radiates over the whole of the vegetative system. The writers mention that Aschner found that puncture of the floor of the third ventricle provoked an acute glycosuria in animals. They explain the absence of glycosuria in their case partly by the fact that the nutritional state of the patient was very unfavourable for its appearance, and partly by the limitation of the neoplasm.

LEONARD J. KIDD.

**A CASE OF INFUNDIBULAR TUMOUR IN A CHILD, CAUSING
(334) DIABETES INSIPIDUS WITH TOLERANCE OF ALCOHOL.**

L. NEWMARK, *Arch. of Internal Med.*, 1917, xix., April, p. 550
(7 figs.).

THE patient was a small, alert, and agile boy of 14, who looked not more than 10 years old: he had grown little, if at all, for a few years previously, and had always been considered small for his years. (His father is only 5 ft. 5 in. tall.) The boy fractured his skull when 4 years old: became addicted to cigarette smoking when 6: and his polyuria and polydipsia began at 9 years of age. He drank alcohol freely, but no one ever saw him drunk. No signs of brain tumour appeared till two weeks before death. The polyuria was continuous during the last five years of life; and although he had headaches at intervals of several weeks, for at least three years, they were not ascribed to

alcohol or any grave organic disease, as they seldom lasted more than a day, and interfered but little with his schooling.

On necropsy, a large vascular tumour was found occupying the region of the infundibulum, extending forward through the lamina terminalis, between the frontal lobes, and backward into the third ventricle, and destroying the posterior lobe of the pituitary, and most of the pars intermedia. The pineal body was atrophied. The writer points out that his case teaches us that in a polyuria resulting from infundibular disease the brain may not be affected by alcoholic draughts to which immaturity, a previous injury to skull, and the presence of a large vascular tumour might be supposed to render it peculiarly sensitive. From a study of recent literature on the tumours of the pituitary and the pineal bodies he concludes that while disease affecting the neuro-hypophysis may provoke polyuria, this effect may not be permanent, and need not occur at all. The polyuria occurs in some cases of pineal tumours. Again, while the neuro-hypophysis has diuretic properties, so has the sheep's pineal body. The writer quotes the case of V. Gierke (1914): a patient of 72 died of metastatic carcinoma of the bones, kidneys, dura, and pineal body: six weeks before death polyuria appeared, and continued to the end.

There were found in the pituitary only traces of a minute hæmorrhage at the posterior border of the anterior lobe, which was not held responsible for the diabetes insipidus. Apart from a slight change in the thyroid, the other endocrine glands were normal, so that V. Gierke was inclined to connect the polyuria with the pineal disease. Newmark, in agreeing with the commonly accepted view that a tumour causing diabetes insipidus is commonly situated in or near the neuro-hypophysis, but occasionally in the pineal body, says that it does not appear from clinico-pathological observations that it is over-production of a diuretic substance that causes diabetes insipidus. (The paper has a large number of interesting references to the literature of the subject, including the experimental studies of Camus and Roussy.)

LEONARD J. KIDD.

GLIOMA OF THE LEFT TEMPORAL LOBE IN A LEFT-HANDED INDIVIDUAL. (Glioma del lobo temporale sinistro in un mancino.) F. GIANNULI, *Riv. di patol. nerv. e ment.*, 1917, xxii, p. 343.

GIANNULI refers to his previous paper on tumours of the temporal lobe (*v. Review*, 1916, xiv., p. 80), and records a case in a previously healthy man who, at the age of 29, began to suffer from headache, auditory and olfactory hallucinations, and convulsive attacks which at first occurred every three months, and then increased in frequency till they became a daily event. Pro-

gressive mental deterioration set in, and death took place seven years after the onset. It is noteworthy that though the patient was left-handed, he always used his right hand for writing. The autopsy showed a glioma infiltrating the left temporal lobe, insula, uncinate fasciculus, antimurus, and putamen.

Giannuli's conclusions are as follows:—

1. Epilepsy possessing the characters of symptomatic or essential epilepsy may be the only clinical expression of a glioma of the temporal lobe.

2. The temporal lobe is one of the most sensitive cerebral epileptogenous zones, as is shown by the frequency with which epilepsy accompanies lesions of this lobe.

3. Left-handedness depends on an inversion of the anatomical asymmetry between the two cerebral hemispheres, which is the cause of right-handedness.

4. Left-handedness may be associated with ability to write with the right hand.

J. D. ROLLESTON.

TUMOURS OF THE GASSERIAN GANGLION. With the report of (336) an operated case. ERNEST SACHS, *Annals of Surgery*, 1917, lxvi., Aug., p. 152 (8 figs.).

OUT of thirty-seven recorded cases of Gasserian ganglion tumours only eight have come to operation, and only two of these were relieved. Two anatomical groups may be made, (1) those containing nerve elements and therefore either growing from elements in the ganglion or including it in their growth, and (2) those growing from the dura covering the ganglion, and compressing it but not involving it. Sachs divides those cases clinically into (1) early, and (2) late cases. "The case here reported also died of a recurrence, but I feel convinced that if certain diagnostic features of these cases are recognised, these cases may come to operation still earlier and have permanent relief." Twenty-one of the thirty-seven cases belong to the early group. Almost all cases have been left-sided.

In December 1914 the patient, a woman, began to have pains in the area of the ophthalmic division of the left trigeminus; two months later it had spread to all three divisions, and was continuous. It was unrelieved by remedies. Paresis of the left abducens, and marked weakness of motor fifth in October 1915. No intracranial signs found. Operation on 27th October 1915. The tumour was very adherent in the region of the ophthalmic branch. In separating this attachment, severe bleeding occurred; two cigarette drains controlled this. Transfusion; quick recovery; but on return to consciousness she had complete third and sixth

nerve palsy, and a complete motor aphasia with slight weakness of right hand. These soon cleared up, but then a left twelfth nerve palsy appeared, with a herpetic blister on the naso-pharyngeal side of the soft palate on the left side. Curiously, after the third nerve palsy cleared up the dilated left pupil became contracted. The operation gave complete left trigeminal anaesthesia and complete relief from pain: patient left hospital about two weeks after operation. Severe pains recurred six or seven weeks later: there was still anaesthesia in left fifth area, but deep pressure sensation was present; the twelfth nerve paralysis was still present. On 12th February 1916, operation again; tumour inoperable. Severe pain, enlargement of cervical glands, extreme emaciation. Death on 15th September 1916. The tumour was regarded as an endothelioma.

Gasserian ganglion tumours have to be diagnosed from (1) true tic douloureux, and (2) sphenoidal sinus disease. In the former the pain is never persistent or continuous: there are periods of relief, and remedies may give temporary relief: there are no cranial nerve palsies. In sinus disease pain may be persistent and of the tumour character, but never, Sachs thinks, as severe; though abducens palsy is a possible complication, motor fifth palsy is not. "Severe continuous pain in the distribution of the trigeminus, with paresis of the motor branch of the fifth nerve, justifies at once the diagnosis of tumour of the Gasserian ganglion. I do not believe it is necessary to wait, however, until the motor branch of the fifth nerve is involved. As soon as an infected sinus has been excluded" (as was done in Sach's case) "the persistent pain, as distinguished from the intermittent spasmodic pain of tic douloureux, justifies an operation on the ganglion on the suspicion that a tumour is present." (There is a bibliography.)

LEONARD J. KIDD.

A CASE OF BASILAR IMPRESSION. (Een geval van basilaire (337) impressie.) H. W. STENVERS, *Nederlandsch Tijdschr. voor Geneeskunde*, 1916, lii., 13 Mei, p. 1733 (6 figs.).

A PECULIAR deformity of the bones of the base of the skull has long been known to anthropologists under the name of basilar impression or plastic deformity. Virchow collected cases among the skulls of the aborigines of Germany in 1876, and the condition is described by Grawitz (*Virchow's Archiv. f. Path. Anat.*, 1880, lxxx., p. 449). It has been theoretically held to be due to osteomalacia, hydrocephalus, or to retardation of ossification; but its cause is still unknown. The case recorded here by Stenvers has the outstanding feature that an adiposo-genital syndrome coincided with an atrophied infundibulum and a

macroscopically and microscopically normal pituitary. His case was a girl of 17, who had never menstruated, and had recently become abnormally fat. Admitted on 20th April 1915, with papillitis. At end of July 1914 severe headaches; in August these increased, and giddiness, vomitings, and epileptoid attacks came on; later, paræsthesiæ in right face and shoulder. In October 1914 diplopia and strabismus; the adiposity had recently appeared. In early November 1914 increase of diplopia. In spring of 1915 she complained of a marked sensation of hunger and tiredness; this increased, and she felt ill; eyes became very prominent. Physical examination on 23rd April 1915—brown discoloration of skin, marked exophthalmos, excessive general adiposity. Left homonymous hemianopia. Temporal fields constricted, especially right. Right field constricted for colours; old neuro-retinitis; bilateral defect of vision not improved. Left pupil, light-reaction feeble; consensual reaction bad from left. Right pupil, feeble convergence-reaction; divergent strabismus; nystagmoid movements, chiefly to left. Right hearing diminished (otitis externa). Tongue comes out to right. Movement of head to left shoulder weak. Breasts moderately developed; pubic and axillary hair absent. Abdominal reflexes normal; plantars flexor; Oppenheim's reflex "dorsal" on right. Later, changes occurred in the visual fields for colours. Cotton-wool sensibility diminished over skin of root areas of L 5, S 1, 2, 3. A skiagram showed changes in region of sphenoidal sinus. On the strength of these signs which pointed to a lesion of, or in the neighbourhood of, the pituitary body, operation was performed; death a few hours later. *Necropsy*.—No trace of a tumour anywhere. The base of skull showed a marked prominence, due to the pushing upwards of atlas and axis, and also of parts of the occipital and temporal bones. There was a furrow on the base of the brain corresponding to this bony deformity. The right trigeminus nerve was thinned; internal hydrocephalus. Floor of infundibulum thinned. Pituitary normal, both macroscopically and microscopically. The thyroid gland showed marked colloid degeneration. Other internal organs normal.

Conclusions.—

1. The adiposo-genital syndrome can exist without any changes in the pituitary body.
2. In the writer's case the adiposo-genital syndrome was accompanied by atrophy of the wall of the infundibulum in the third ventricle.
3. From the evidence of other observations it is probable that there are important centres in the infundibulum for the innervation of the autonomic system.

4. It is possible that the adiposo-genital syndrome is due to the destruction of this infundibular autonomic centre.

5. So long as the origin of the atrophy of the walls of the third ventricle is unknown, it is an open question whether this is due to the basilar impression, or whether the basilar impression is itself due to the trophic disturbances.

LEONARD J. KIDD.

A CASE OF CEREBRAL TUMOUR WITH TUMOUR OF THE (338) SKULL. JOHN TATTERSALL, *Journ. Ment. Sci.*, 1917, April, p. 250.

FOLLOWING a trauma a young man developed optic neuritis proceeding to complete atrophy with, latterly, headache and vomiting accompanied by mental facility and amnesia for recent events and dates. He showed a tendency to walk to the right, conjugate deviation of the eyes to the right, nystagmus on turning his eyes to the right, and he held his head to the right. All other nervous symptoms were negative. A swelling appeared later on the right side of the skull. He died suddenly after an illness of ten years. The post mortem revealed a tumour whose centre occupied the whole width of the right cerebral hemisphere, sparing the frontal and occipital lobes, but encroaching on the lateral ventricles. The skull over this region also exhibited tumour growth. The tumours were not in contact and microscopically proved to be both endotheliomas.

The author comments on the absence of localising signs, considering that "all the fibres of the corona radiata must have been cut off." (Skiagrams and photographs are appended.)

H. DE M. ALEXANDER.

TUBERCLES OF THE PONS. (*Tubercules de la protubérance.*) AD. (339) D'ESPINE and V. DEMOLE, *Arch. de Méd. des Enfants*, 1917, xx., Juillet, p. 355.

A BOY of 7, whose sister had died of intestinal tuberculosis at the age of 8 years, was treated for scarlatina: the lungs showed no physical signs, and the cuti-reaction was negative. Six months later, after perfect health, vertical headaches with loss of appetite appeared. He had paralysis of conjugate movement of the eyes to the right and paralysis of the right facial nerve. Otherwise he had a general absence of visceral signs, with the exception of definite bronchophony at the level of the first dorsal vertebra (D'Espine's sign) which denotes the presence of bronchial adenopathy. Occasionally the boy could, by a violent effort, make his left eye look slightly to the right of the middle line. Occasional diplopia for objects to his right. Otological

examination negative. Perfect movements of limbs, but very exaggerated knee jerks, with bilateral ankle clonus, but no Babinski sign. Cuti-reaction now markedly positive. Occasional slight elevation of temperature towards evening. The diagnosis was a solitary tubercle of the right eminentia teres: at the same time there appeared to be a pontine lesion which was irritating the pyramidal tracts. The boy improved and went home in June 1915. For three months this improvement continued, though many of his physical signs were still present. The boy put on weight and returned to school. On 19th October alimentary vomiting, constipation, and fever. Signs pointing to tubercular meningitis appeared. Early in November the boy became worse, could not walk or sit; Kernig's sign increased in degree: delirium, and incontinence of urine: a slight flaccid palsy of left arm. Bilateral papilloedema. Death on 7th November 1915. Necropsy showed tracheo-bronchitis, caseous pulmonary tuberculosis, caseation of tracheo-bronchial lymph-nodes, tubercular ulceration of intestines, multiple conglomerate tubercles of cerebrum, cerebellum, and bulb, recent miliary tuberculosis, and acute tubercular meningitis. The cerebral ventricles were dilated. There were fourteen small, firm, yellowish tubercles on the corpus callosum and in the cerebral and cerebellar cortex. On the floor of the fourth ventricle, exactly on the right eminentia teres and extending thence for 2 or 3 mm. by the side of the fovea superior, was a solitary tubercle, 6 mm. in diameter, regularly circular, slightly prominent, sub-ependymal, and covered by fine venous arborisations. There was oedema of the cerebral substance, especially on the right. Histological examination showed that the right sixth nucleus was almost entirely destroyed (there is no mention that the left third nerve was examined): the right facial nerve was almost entirely degenerated: the outer border of the right dorsal longitudinal bundle was similarly degenerated. In the centre of the right pyramidal tract in the pons there was a solitary tubercle, $2\frac{1}{2}$ mm. in diameter. Thus, the first tubercle in the right eminentia teres had destroyed the right sixth nucleus and severely damaged the right facial nerve: the second had separated the fibres of the right pyramidal tract without seriously injuring them. This explains the absence of hemiplegia, and the exaggerated jerks and clonus. LEONARD J. KIDD.

ADIPOSO-GENITAL DYSTROPHY WITH MYOPATHY. (*Dystrophia* (340) *adiposo-genitalis mit myopathie.*) SIGISMUND V. DZIEMBOWSKI, *Deutsch. Med. Wochschr.*, 1917, xliii., May 24, p. 654.

A MECHANICAL dentist, aged 26, presented an adiposo-genital syndrome with myopathy. Many of his family were fat or

neurotic. His right hand was injured at his birth, which was prolonged and difficult. During childhood health good, but right hand weak; cramp and stiffness often occurred in the right fingers. The right leg was often stiff in walking. In his fifteenth year he had very severe headaches, and then he quickly became very fat. The adiposity increased greatly on the face, shoulders, thorax, and belly; at times this was so rapid that the skin and its vessels burst, with bleeding. The adiposity troubled him for three years, and then became stationary. Then appeared polydipsia and polyuria. Then, at 25, he noticed his testes were smaller than formerly, and erections and emissions became less frequent, and sexual desire weaker. During the last few months before admission increased weakness, especially on right half of body. Hair on face and thorax scanty, on pubes of feminine type; pelvis and hands feminine. Bitemporal hemianopia; other sense-organs normal; thyroid gland normal. Heart a little dilated; blood pressure low; lymphocytosis; sugar tolerance much increased. Internal viscera normal. A skiagram showed changes in sella turcica. The myopathy was very marked, especially on the right side of body. A piece of biceps muscle excised showed macroscopically excess of fat, and microscopical examination showed that the scanty muscle-bundles were markedly atrophied; they are surrounded by very abundant adipose tissue. He was given injections of pituitrin and hypophysin twice daily. After fourteen days there was a most gratifying result: weight fell $4\frac{1}{2}$ kilos., lymphocytosis disappeared, blood pressure rose, and the increased sugar tolerance quickly diminished; the sexual functions showed daily improvement. With all this objective improvement there was marked subjective improvement, increase of strength, loss of tired feeling in his limbs, and of his feeling of deadness. An increase of muscle tonus occurred. The muscular movements became stronger, especially in the hand-grips. Improvement was very noticeable in the facial musculature, and the electrical reactions everywhere improved. The heart muscle shared in the improvement, pulse became much slower, and the increase of pulse frequency after slight effort also disappeared. Treatment by extracts of thyroid, adrenals, and testes had no effect on the patient's condition. The writer regards the myopathy as undoubtedly a result of the pituitary hypofunction; the improvement in the symptoms of the adiposo-genital syndrome went hand in hand with that of the myopathy. He concludes that the pituitary secretion, supplied therapeutically, either acted directly on the musculature or activated the secretions of other glands.

LEONARD J. KIDD.

DEATH FOLLOWING THE INTRAVENOUS INJECTION OF
(341) **SALVARSAN.** M. D. WANDIA, *Ind. Med. Gaz.*, 1916, li., p. 388.

A SEPOY, aged 23, suffering from secondary syphilis, was given an intravenous injection of 0.5 gm. salvarsan at 10 A.M. During the night he had much vomiting and diarrhoea. On the sixth day he became deeply jaundiced. The liver was enlarged, and there was no leucin or tyrosin in the urine. Acute yellow atrophy was thus excluded. Death took place on the tenth day. No autopsy.

J. D. ROLLESTON.

NINHYDRIN REACTION IN THE EXAMINATION OF CEREBRO-
(342) **SPINAL FLUID.** N. NOVICK, *Journ. Infect. Dis.*, 1917, xxi., July, pp. 52-55.

THIS reaction was described by Noble in 1915. It consists in shaking and boiling for a few seconds 0.1 c.c. of a watery solution of 1 per cent. ninhydrin with 0.5 c.c. of spinal fluid. On cooling, a purple colour, turning into a deep blue, indicates a positive reaction. A negative reaction gives either a straw-yellow colour or no colour at all. Noble asserted it was of value in differentiating tuberculous meningitis from other diseases in which a clear fluid was yielded.

The author found the reaction is of moderate differential diagnostic value. It runs, as nearly as may be marked, parallel with the albumin and globulin content of a given fluid. It may be used as a bedside presumptive test, but has no advantage over the albumin and globulin test, and is incomplete without the cytologic and bacteriologic examination, as it does not separate the non-purulent pathologic fluids.

A. NINIAN BRUCE.

XANTHOCHROMIA AND OTHER CHANGES IN THE CEREBRO-
(343) **SPINAL FLUID: THEIR FREQUENCY IN CHRONIC**
SURGICAL SPINAL DISEASES, AND THEIR SIGNIFI-
CANCE. CHARLES A. ELSBERG and EDWARD L. ROCHFORD, *Journ. Amer. Med. Assoc.*, 1917, lxxviii., June 16, p. 1802.

THIS study is based on an analysis of the cerebro-spinal fluids of ninety-two patients suffering from chronic spinal diseases who were operated on by Elsberg; in all the patients the diagnosis was substantiated or corrected by the operative findings and by the examination of material removed at the operation, and in a few cases at necropsy. The writers note that xanthochromia was not observed in any of the twelve cases of intramedullary growth or in any case of extradural neoplasm. Conclusions:—

(1) Xanthochromia is strongly suggestive of spinal cord tumour in lower dorsal and lumbar regions.

(2) The combination of yellowness, high protein content, spontaneous coagulation, and pleocytosis (Froin's syndrome) is characteristic of the large endotheliomas or sarcomas which surround the conus and roots of the cauda equina.

(3) Nonne's syndrome (increase of globulin, without increase of cells) is suggestive of extramedullary spinal cord tumour.

(4) Increase of globulin without increase of cells, plus yellow discoloration of the fluid, make the diagnosis of extramedullary spinal cord tumour very probable.

(5) The changes in the fluid may be a valuable aid for the differentiation between spinal diseases in which operative interference may be necessary.

(6) The diagnosis of a spinal disease should never be made from the spinal fluid findings alone; the results of the examination of the fluid obtained by lumbar puncture should be used only as a diagnostic aid.

The writers mention that, as none of their patients with multiple sclerosis had an excess of globulin in their fluid, this absence of globulin may be a useful diagnostic aid in the differentiation between that disease and spinal cord tumour in the occasional patient in whom the diagnosis is difficult.

LEONARD J. KIDD.

THE SIGNIFICANCE OF XANTHOCHROMIA OF THE CEREBRO-

(344) SPINAL FLUID. T. P. SPRUNT and J. E. WALKER, *Bull. Johns Hopkins Hosp.*, 1917, xxviii., Feb., p. 80.

A CASE CONFIRMATORY OF THE SPINAL FLUID COMPRESSION

(345) SYNDROME. CHARLES W. HITCHCOCK, *Journ. Amer. Med. Assoc.*, 1917, lxviii., May 19, p. 1474.

SPRUNT and Walker's paper is based on five cases observed and reported by them, and an analysis of 100 cases from the literature. By the name of "Froin's syndrome" we understand several peculiarities of the cerebro-spinal fluid which Froin described in the fluid from a patient suffering from an organic nervous disease, viz., a yellow colour, an increased number of lymphocytes, and a marked and rapid coagulation. By the term "Nonne's syndrome" is meant the presence of globulin in large amount without cell increase in the cerebro-spinal fluid. Of this latter syndrome Sprunt and Walker say it "has apparently much the same significance as Froin's syndrome. Perhaps, as Hanes suggests, they are merely different stages of the same process, the xanthochromia appearing late; or, as Nonne believes, they may be variants due to the presence or absence of blood." They further remark that in different cases there are variations in the

amount and rapidity of clot formation, in the presence or absence of lymphocytosis, and in the amount of globulin. Owing to the difference in points of view or in methods of examination certain observers, in describing what is probably the same type of fluid, emphasise some of these characteristics and neglect to mention others. In general the Germans are interested in the large amounts of globulin—the positive phase 1 of Nonne—and frequently say nothing of coagulation, whereas the French lay stress upon the coagulation *en masse* and may make no mention of the presence or absence of globulin or cells. “Hence we feel that in tabulating cases for study we should not make the criteria too strict nor attempt to conform too rigidly to the description of any one author, as Mix seems to have done in insisting upon massive coagulation as a necessary feature of the syndrome.”

In summarising the results of their studies the writers divide the fluids into two main groups:—

1. Those in which the colour is due to dissolved hæmoglobin or its derivatives, and which as a rule do not coagulate spontaneously and contain only a small amount of globulin. Such fluids are associated with brain tumours in contact with the meninges or ventricles.

2. The larger and more important group comprises those cases showing the so-called Froin's syndrome, in which the fluid is transparently clear, yellow, coagulates spontaneously, contains large amounts of globulin, may or may not show pleocytosis, and gives no positive tests for hæmoglobin.

This is a “compression syndrome,” its main determinants being the isolation of a lumbar cul-de-sac, in which the spinal fluid stagnates, and probably some vascular changes within its walls. Clinically, with negative X-ray of the vertebral column, it is strongly suggestive of a tumour of the spinal cord, although it may also be associated with intradural inflammatory processes.

(In the first case reported by the writers the Froin compression syndrome was of direct importance in enabling them to arrive at the correct diagnosis. They mention that Oppenheim reported two cases in which this finding at lumbar puncture clinched the diagnosis of spinal cord tumour, for which, without this finding, there was insufficient evidence. And Raven pointed out that, while this syndrome is evidence of compression, it tells nothing concerning the nature or the site of the lesion.)

The case reported by Hitchcock was a man of 23, who walked into hospital without noticeable abnormality of gait. He complained of pain in abdomen and back, of ten days' duration, which largely disappeared after characterisation had relieved the enormously distended bladder. He said he had twice before had

retention of urine some years before. Admitted on 20th November: recent loss of flesh. Marked rigidity of neck: horizontal nystagmus on looking down or to left. On 21st November right knee jerk exaggerated, left diminished: feeble abdominal reflexes, cremasterics present: no apparent sensory changes, some cervical rigidity, but no Kernig. On 22nd November deep reflexes abolished, complete flaccid palsy of both lower extremities. This progressed to total abolition of all reflexes and an ascending sensory impairment, analgesia being for several days stationary, up to level of 12th dorsal vertebra. On 21st November the spinal fluid was clear, but clotted rapidly. On 23rd November it flowed very slowly and clotted instantly. On 27th November spontaneous coagulation, well-marked xanthochromia, and no pleocytosis. These changes led to diagnosis of a compression myelitis and the advice of surgical interference. On 2nd December laminectomy was done, with free escape of (extradural) pus at the level of the 7th dorsal vertebra, revealing an osteomyelitis of that vertebra as the source of trouble. Death on 10th December: no necropsy. "The case, however, well illustrates the presence of the Nonne-Froin syndrome in cases of compression myelitis."

LEONARD J. KIDD.

AUDITORY DISTURBANCES AND EPILEPSY. (*Troubles auditifs (346) et épilepsie.*) RIMAUD, *Rev. de Laryng., d'Otol., et de Rhinol.*, 1916, xxxvii., p. 164.

THE writer records a case of what he regards as a curious case of epilepsy. Patient is a mason, aged 25, alcoholic. Grandfather alcoholic, several attacks of delirium tremens. An uncle insane. Family history otherwise unimportant, except that one brother died from meningitis after a month's illness, aged 16. Patient had infantile convulsions at 3 years and again at 7 years. Nocturnal enuresis up to 13. Good general health; no infectious diseases; denies syphilis. He now complains of attacks of temporary deafness, with permanent and progressive diminution of hearing. These attacks began about the age of 6 years. They are of sudden onset, without known cause, and last for some minutes up to a maximum of fifteen or more during which time he does not hear what is said near him. At first the attacks occurred only at some weeks' intervals; they have gradually become more frequent, but have been absent during his recent two years of military service. During the past few months attacks more frequent; he may go a week without one, but may have four or five a day; his hearing becomes progressively less acute. At the beginning of attacks he has tinnitus in both ears, like the sound of water; instantly he fails to hear

what those near him are saying; he believes that this complete deafness lasts one or two minutes; during this time the tinnitus gradually lessens. Hearing returns to his normal rather quickly, and always without vertigo. There is no falling, no cry, no involuntary micturition, and no convulsion. Physical examination is negative except for definite diminution of hearing, but without any objective lesion. Perosseal hearing diminished. He is irritable and gloomy, and is disturbed that "he is not like others." He is taciturn, avoids his parents, has frequent nightmares (alcoholism) difficulty in attention; sexual frigidity, masturbation. He was treated for three weeks by 3 gm. of bromide daily; the attacks became less frequent. His parents state that his attacks last for ten minutes, not merely one or two as he thinks. Probably he really loses consciousness during attacks. The writer discusses at great length the relation between aural affections and epilepsy. He concludes that there is a rather rare auricular form of epilepsy.

LEONARD J. KIDD.

THE PSYCHOLOGY OF HYSTERIA. W. G. SOMERVILLE, *Amer. Journ.* (347) *Insan.*, 1917, April, No. 4, p. 639.

HYSTERIA is described as a functional nervous state closely related to hypnotism, characterised by various peculiar symptoms and phenomena, and dependent on suggestions either auto or external. The symptoms of the hysteria are the expressions of dissociated or submerged wishes, and are unconscious means for the attainment or fulfilment of a wish. In discussing the difficulty of distinguishing between hysteria and malingering, it is pointed out that the symptoms produced by the subconscious are maintained without effort, and for a longer period than those brought about consciously, that in the former the symptoms continue even when not observed, while in the latter they may disappear when there is no fear of detection.

Nine interesting cases are reported. D. K. HENDERSON.

THE GLUTEAL SIGN IN SCIATIC NEURALGIA. (Le signe du fessier dans la névralgie sciatique.) FÉLIX ROSE, *Presse Médicale*, 1917, Ann. xxv., 4 Juin, p. 319.

THE patient is placed flat on his face and is told to relax his gluteal muscles. With a percussion hammer a light stroke is given at the points of insertion of the gluteus maximus on the border of the second, third, and fourth pieces of the sacrum, first on one side and then on the other. In the large majority of cases of true sciatic neuralgia, and also of cases of traumatic irritation of the nerve, there is produced a fascicular contraction of the

muscle under the skin which is quite different from a voluntary contraction, which is global. Sometimes this exaggerated gluteal reflex occurs only on percussion of the affected side, at others equally on percussion of the sound side. The writer thinks that it is probably present also in some cases of sacro-iliac arthritis, and possibly also of hip disease or arthritis. LEONARD J. KIDD.

**NOTE ON THE TREATMENT OF NEURITIS, WITH SPECIAL
(349) REFERENCE TO SCIATICA.** HARRINGTON SAINSBURY, *Lancet*,
1917, excii., June, p. 111.

THE author applied (in cases of sciatica) strong hydrochloric acid of the B.P. (half a drachm to a drachm sucked up into a wad of cotton wool the size of the knuckle of the middle finger and held between the previously vaselined finger and thumb) "in a broad line some $1\frac{1}{2}$ in. across, straight down the back of the thigh from the gluteal fold to the popliteal space; another application of like breadth for about 3 in. behind the head of the fibula along the oblique course of the external popliteal nerve at this spot; lastly, for some 5-6 in. behind the external malleolus obliquely on to the dorsum of the foot." The extent of the tenderness—such as Gowers' spots—is the guide. Two or three applications may be made at one sitting if the skin bears the acid well—but gauge this by applying the first coat lightly. It causes little discomfort. A papular rash may appear in delicate skins. Blistering has not occurred. Repeat the treatment—a camel-hair brush may be used instead of the cotton wool—twice a week or oftener so long as the pain and tenderness continue, and supplement with massage.

A large experience has shown the clinical value of this acid in sciatic neuritis and myalgia. Striking results were obtained in three recent cases in a military hospital—two of them being bad cases of long duration.

The explanation of the action of the acid in these cases is unknown. (The treatment was recommended to the author by Dr Hugh Wingfield.)

H. DE M. ALEXANDER.

**A CASE OF UNILATERAL VERTICAL NYSTAGMUS ACQUIRED
(350) IN ADOLESCENCE, AND CAUSED BY AN ACCIDENT.**

ASTON LUTZ, *Archives of Ophthalmol.*, 1917, xlv., July, pp. 357-362.

UNILATERAL nystagmus is a rare condition. This case occurred in a woman who received some brick dust in her left eye when 13 years old. When the bandage was removed she noticed all objects dancing before her left eye. This had continued up till the present time, when she was aged 39.

Examination showed that the patient in her earliest life suffered from a severe inflammation of the optic nerves, which left her with both optic nerves atrophic, diminished vision, and a central scotoma for colours in the right eye. This was responsible for a slight, nearly imperceptible nystagmus of the right eye. The traumatism in her thirteenth year produced a corneal opacity in the left eye, still further reducing the left vision. This new injury apparently provoked a unilateral nystagmus in predisposed eyes. It is remarkable that no divergent strabismus developed during these twenty-six years.

A. NINIAN BRUCE.

A NEW TREATMENT FOR PARALYSIS AGITANS. WALTER B. (351) SWIFT, *Journ. of Amer. Med. Assoc.*, 1916, Dec. 16.

A MARKED case of paralysis agitans was treated with slow moving exercises taken for fifteen minutes three times a day, and resulted in (1) a general constant relief of bad feelings, (2) occasionally an hour of complete relief from all distressing symptoms, and (3) a quickly gained repose into sleep on retiring.

The treatment, though giving marked relief from intense suffering, even to the point of recovering normal conditions for hours at a time, proves no cure, and the symptoms all returned when the exercises were omitted; but they showed improvement again when resumed, and also again gave relief.

AUTHOR'S ABSTRACT.

WHAT IS A "SPEECH CIRCLE"? WALTER B. SWIFT, *Journ. of Nat. (352) Education Assoc.*, 1917, April.

To summarise, the speech circle consists of three individuals with three functions—superintendent, doctor, and teacher; supervision, diagnosis, and treatment.

AUTHOR'S ABSTRACT.

REMARKS UPON THE VEGETATIVE NERVOUS SYSTEM AND (353) THE INTERNAL SECRETIONS. FREDERICK J. FARNELL, *Journ. Ment. Sci.*, April 1917, p. 225.

THIS paper is a summary of current views, and on account of its condensation it does not lend itself to abstraction.

H. DE M. ALEXANDER.

VIRILISM—FORME FRUSTE. HENRY K. MARKS, *Journ. Nerv. and (354) Ment. Disease*, 1917, xlv., July, p. 17.

IN this preliminary report Marks discusses the question whether a form of virilism fruste exists. He gives details of twenty-two unselected cases of women who all showed hirsutism of male type:

many had menstrual irregularities, others sterility, others sexual frigidity, and yet others homosexuality. He claims that he has traced a series of clinical pictures in the female, the central point of which is the striving towards maleness, with corresponding reduction or loss of female characteristics. The evolution of the syndrome has been followed from the pseudo-hermaphroditism of the embryonal period to the so-called virilism of adult life. In all the various forms the clinical result has been the formation of sexually intermediate types, the pathological findings binding them together, tumour or hyperplasia of the interrenal tissue. On the basis of his twenty-two observations, Marks claims that a type of virilism exists which corresponds essentially to the clinical picture of the "virilisme surrénale" of Gallais (*v. Review*, 1912, x., p. 345); but which is attenuated and compatible with life—in a word, a "Virilisme forme fruste." He writes: "Granted the existence of virilism forme fruste, it is apparent that an interesting field of inquiry is opened up from a sociological and eugenic point of view; likewise questions that touch upon the 'infantile trauma' of Freud in the origin of homosexual traits."

LEONARD J. KIDD.

HYPERTHYROIDOSIS ASSOCIATED WITH GYNECOMASTIA.

(355) J. R. FREEMAN, *Therap. Gaz.*, 1916, xl., p. 9.

THE patient was a man, aged 37, the father of four children. His sister, one of his daughters, and a daughter of his first cousin also had a goitre. The abnormal development of the mammae was noted three months after the nervous symptoms appeared.

No previous case of this association has been recorded.

J. D. ROLLESTON.

PSYCHIATRY.

DR HUGHLINGS JACKSON ON MENTAL DISORDERS. Sir GEORGE

(356) SAVAGE, *Journ. Ment. Sci.*, 1917, lxiii., July, p. 315.

THE author has long considered that the relationship of Dr Jackson's teaching in reference to nervous disorders has not been sufficiently considered from the psychiatric side.

Jackson was an evolutionist, and his creed may thus be given:—"Mental operations are simply the subjective accompaniments of sensori-motor processes. The incentives to volition are sensations received through the organs of sense, or the revived impressions of such sensations. The sensori-motor apparatus of the cortex is re-represented in the higher centres, *i.e.*, the prefrontal region

(non-excitabile), where they have the power of controlling and concentrating consciousness in definite directions, and deciding between courses of action." The organs of sense in their relation with their environment are represented in the brain in three grades, ranging from the automatic to the reasoned.

States of consciousness or mind are different from nervous states of the highest centres. Although they occur together, there is no interference of one with the other, hence psychical things are not functions of the brain, but occur during the functioning of the brain. In a sense the whole body is the organ of mind, and Lewis considered that some degree of consciousness attends activities of even the lowest centres.

Jackson agreed with Mercier's views on the difference between unsoundness of mind and so-called insanity, and he placed among the insanities a good many states of mind that are not generally considered included in the insanities. He asserted there were four factors in insanities (Jackson preferred the term insanities rather than insanity): the different depths of dissolution (there being three grades, the degree of mental disorder being related to the amount of dissolution of the highest nervous centre); the person who has undergone dissolution; the rate at which dissolution has been effected; and lastly, the influence of bodily states and external circumstances.

The lowest level does the menial work; the highest level, evolved out of it, becomes practically independent of it, and is the anatomical basis of mind; *e.g.*, what in the lowest levels are centres of the simple reflex actions of eyes and hands are evolved in the highest centres into the physical basis of visual and effectual ideas.

In post-epileptic insanities the dissolution is local in the sense that it preponderates in the highest centres of one-half of the brain, and the mania following a fit is the outcome of activities on the levels of evolution remaining. Dissolution is rarely uniform.

There are three grades of dissolution in epilepsy—the aura, the convulsions, and the state of unconsciousness; Jackson contended all these states were scientifically—not clinically—insanities.

Jackson, in writing on mental disorder, frequently speaks of the perfectly normal physiological action of the parts that have been released from control, and asserts that the same relaxing of control does not necessitate any pathological change in the parts then acting. "The physical condition of those positive mental symptoms (an insane man's illusions and delusions) is not caused by a pathological diseased process. Disease is, I submit, answerable only for the co-existing negative element of insanity."

H. DE M. ALEXANDER.

**A CASE OF SYSTEMATISED DELIRIUM OF PERSECUTION
(357) WITH PSYCHO-SENSORY HALLUCINATIONS. P. M.**

TOLEDO, *Journ. Ment. Sci.*, 1917, April, p. 258.

A WELL-EDUCATED paranoiac of 38 years explained his morbid sensations ("internal sensations," later becoming "something crawling in his abdomen," "teeth gnawing his ribs") and hallucinations (people making remarks about him in the streets, voices speaking in his abdomen) as follows:—"On my conception, three ovules were impregnated; but instead of triplets my mother gave birth to me alone and by a sort of an *embryonic aberration*" (patient had read a book on embryology) "the two other fecundated ovules developed into two ape-like beings living parasitically in my inside. They—a male and a female—hate me and are the cause of my internal sufferings. They insult passers-by by words and sounds, and the people look angrily at me."

The patient pestered surgeons to operate on him, and one incised the skin on both sides of his abdomen under a general anæsthetic, suturing the wounds. The patient was assured there was nothing abnormal in his abdomen, but now thinks the surgeon is an "ally" of his two internal enemies.

In ordinary conversation the patient is quite sensible, but occasionally slaps his abdomen to keep his enemies quiet, and has spent a large sum of money visiting cities in search of a surgeon to release him from them. He is not confined, his relatives considering him a neurasthenic.

H. DE M. ALEXANDER.

**HALLUCINATIONS IN THE SANE. ROBERT HUNTER STEEN, *Journ.*
(358) *Ment. Sci.*, 1917, lxiii., July, p. 328.**

THE author gives numerous examples of hallucinations occurring under the following classification:—

(A) Hallucinations the result of causes operating upon the brain or nerves—

1. Toxins—(a) exogenous, *e.g.*, drugs; (b) endogenous, *e.g.*, fever, gout.
2. Disorders of the cerebral circulation; *e.g.*, anæmia, congestion from apoplexy, asphyxia.
3. Disease of end-organs; *e.g.*, retinal hæmorrhage.
4. After-images.
5. Brain diseases of obscure pathology; *e.g.*, epilepsy, migraine.

(B) Hallucinations of mental origin—

1. Suggestion (the main element being expectant attention).
2. Hypnotism.
3. Crystal gazing, clairvoyance, and clairaudience.
4. Hysteria, somnambulism, multiple personality.
5. Hypnagogic visions (generally in youth).
6. Dreams.
7. Hallucinations in history.
8. Collective hallucinations (the angels at Mons).
9. So-called telepathy.
10. Hallucinations the result of a complex.

Hallucinations *do* occur in the sane, and do not differ from those occurring in the insane, and the author thinks such cases should be extensively studied, especially in border-land or hysterical cases. As toxins can produce hallucinations in the sane, a similar origin is probably the cause in some cases of insanity, and they occur most frequently in alcoholic insanity. Hallucinations can occur independently of physical changes, as an idea may be visualised or converted into a voice, smell, or other sensory phenomenon, and may be introduced from without or arise from within; and in certain cases hallucinations in the insane can be best studied from a psychological rather than from a physiological point of view. Some process of dissociation is probably at work in every case presenting hallucinations, as they occur in cases of multiple personality where dissociation is seen in greatest extent.

H. DE M. ALEXANDER.

THE MANAGEMENT OF CONFUSIONAL STATES, WITH SPECIAL

(359) **REFERENCE TO PATHOGENESIS.** A. WILLIAMS, *Journ. Ment. Sci.*, 1917, lxiii., July, p. 389.

CONFUSION is the hall-mark of the effects of toxin upon the cerebrum. Interference with neuronal conductivity is the chief pathogenetic factor, and the locality of this disturbance is one of the determinants of the form taken by the psychosis, whether hallucinatory, disorientative, depressive, delusional, &c. Another factor is the state of the body secretions as affected by the toxins.

Treatment should aim at combating the aetiological factor of the confusion by diminution of the nitrogenous food when the kidney or liver is at fault, by correcting the proper adjustment of the carbohydrate intake where symptoms of acidosis occur, and by psychotherapy where psychological factors are at work. Physiological irritability must be counteracted by hydrotherapy, fresh air, and non-stimulating food.

A few cases are detailed illustrating the methods advocated.

H. DE M. ALEXANDER.

ZOLA'S STUDIES IN MENTAL DISEASES. J. BARFIELD ADAMS, (360) *Journ. Ment. Sci.*, 1917, April, p. 165.

As a rule novelists are not successful in their delineation of the insane—they overdo it.

Zola, however, studied human beings as a naturalist and scientist, consequently his pictures of health and disease are drawn with a truth—more particularly in his later novels—which is rarely found in the works of other novelists, and in his analysis of the mind he does not forget the body. His pictures of mental disease are interesting, because his characters portray their disease in the midst of their everyday surroundings in civil life.

In “*La Couquète de Plassans*,” François Mouret was a manic-depressive, and Marthe Mouret was phthisical, erotic, cataleptic, and hysterical.

In *Silvère Mouret*, *Serge Mouret*, *Lazare Chanteau*, and *Claude Lantier*, Zola portrays four adolescent neurotics.

The mental and physical decadence, the attacks of delirium tremens, in the drunkard *Coupeau Macquart* in “*L'Assommoir*,” are drawn with a realistic pencil, though Zola, contrary to general opinion, states that hard drinking destroys the passion of jealousy.

Homicidal impulse with sexual perversion is illustrated in the case of young *Jacques Lantier*, together with the overwhelming sense of relief, the absence of remorse, the semi-amnesia following the committal of the deed (murder).

Désirée Mouret, *Charles Saccard*, and the foundling *Marjolin* are three imbeciles varying in their mental equipment and impulses.

Apart from insanity Zola also shows in his novels the mental disturbance ensuing in normal people as the result of fatigue and starvation, and he describes the delirium of sickness and of approaching death.

H. DE M. ALEXANDER.

Reviews

AN INDEX OF SYMPTOMS, with diagnostic methods. RALPH W. (361) LEFTWICH. Sixth Edition. Pp. xii+555. Smith Elder & Co., London. 1917. Pr. 10s. net.

DIAGNOSIS is the art of identifying a disease by collating and studying its symptoms. Hitherto the principal classification of these has been the division into subjective and objective symptoms. Patients, according to the author, may be divided into four classes—the pessimist, the optimist, the complaisant, and the mendacious. The pessimist is usually hysterical or “liverish,” the optimist does not mention half his complaints, the complaisant patient answers according to what he imagines is in the mind of the questioner, while the mendacious patient, although not wilfully untruthful like the malingerer, by exaggeration, misrepresentation, and omission, gives an entirely misleading conception of his symptoms.

In order to aid in the interpretation of symptoms, a complete catalogue is here given of every symptom, and the numerous different diseases in which it may be present, *e.g.*, under the heading “taste” all the diseases are given associated with (1) lost or impaired taste (ageusia); (2) hemiageusia; (3) perverted taste (parageusia); and (4) foul taste (cacogeusia). The nails are considered under the headings of cyanosed nails, brittle nails, nails shed, incurved nails, grooved nails, enlarged and thickened nails, ulcers round nails, koilonychia, “reedy” nails, and Quincke’s sign. Similar compilations of diseases are given for every symptom, with a high degree of exhaustiveness. The word “symptom” is taken in its broadest sense, and includes every factor in the diagnosis, and no distinction is thus drawn between medicine and surgery. A section has been added containing brief descriptions of some seventy more or less rare diseases, while another section gives the different synonyms for different diseases, and still another section termed “eponymous signs” gives the names of individuals associated with different diseases. As a work of reference such a scheme is undoubtedly of use, and the fact that this book has now reached its sixth edition, shows that such compilations, which must originally have entailed much work, have proved of considerable help to practitioners.

COLLECTED PAPERS ON ANALYTICAL PSYCHOLOGY. By C. G. (362) JUNG, M.D., LL.D. Authorised translation edited by Dr Constance E. Long. Second Edition. Pp. xxviii+492, with 1 coloured plate and 15 figs. in text. Baillière, Tindall, & Cox, London. Pr. 15s. net.

WE have already reviewed the first edition of this book (*v. Review*, 1916, xiv., p. 412), and note with satisfaction that a number of improvements suggested by us then have been incorporated in this edition, *e.g.*, the papers are published as far as possible in chronological order, and the names of the different translators are given in footnotes at the beginning of each essay.

A new chapter has been added upon "The Concept of the Unconscious," being a lecture delivered by Dr Jung early in 1916 before the Zürich Union for Analytical Psychology. The chapter upon the psychology of the unconscious processes has been fundamentally altered, and the results of more recent researches included. These additions still further enable the reader to become familiar with the recent views of the Zürich School, and at the same time to understand how they differ from the Freudian standpoint.

THE PSYCHOLOGY OF SOUND. HENRY J. WATT, M.A., Ph.D., (363) D.Phil. Pp. viii + 241. Cambridge, at the University Press. 1917. Pr. 10s. 6d. net.

THE aim and end of the study of hearing is to explain it. This requires first a statement of the facts of hearing and their connections. These are wholly and solely matters of experience, and are thus psychical. At the present time even the primary part of the study of hearing is full of the keenest disputes on account of the peculiar complexity of even the simplest auditory experiences, and the difficulty of finding a proper classification. It is also clear that the psychology of hearing must be in harmony with the physiology of the ear, and that a comparative psychological study of the other senses will be of help, as we must expect similar parts of experience, *i.e.*, the various senses, to work in essentially the same way. "Thus there comes into view a 'pure' science of experience as a perfectly homogeneous, closed system of reality without prejudice to its dependence on other systems of reality, and to the particular changes and rules thus forced upon it."

The volumic nature of sounds leads us to recognise that tones are not atoms, but rather molecules of auditory sensation, the smallest masses we find before us. Noises are auditory masses that are characterised by extreme departure from the regularity and balance of tones. This irregularity of mass is attainable by various means, especially by the rapid oscillation of vibratory

rates, by which means the dominance of pitch is more or less obliterated. Pitch appears as the intensively predominant atom of the whole volume which is otherwise regularly and symmetrically balanced in relation to pitch. Tones and noises are not reducible to one another, but to a common "atom" of auditory sensation, or particles of experience, in just as true a sense as water consists of particles of oxygen and hydrogen.

The analysis and theory of hearing put forward in this monograph is thus a purely psychological one. The author's aim here is to expound and to prove his own theory, not to review and apportion the history and merits of all others. The subject is complicated and involved, but the author endeavours to explain himself by two successive summaries and an untechnical account for the benefit of those unaccustomed to psychological terminology, in which special attention is paid to the musical issues of hearing. He emphasises the fact that, in his opinion, the view that the visual parallel to the pitch differences of musical tones is the series of colours in the spectrum is entirely wrong. Music, as an art, is more like the arts of etching or drawing, which dispense with all change of colour, except the series from white to black. Sounds are not the parallel of colours.

The present monograph upon the psychology of hearing is only an episode in a larger effort. The author firmly believes that all our sensory experiences can be completely accounted for in general systematic terms without having recourse to the discoveries of physics or physiology at all, and that "sensory experience would appear as a perfectly self-complete, closed system of reality."

There is no doubt about the value of this book, and it is to be hoped that as the author matures and extends his views, he will present us with further contributions not only to the psychology of hearing, but also to the psychology of the other senses.

ORGANIC TO HUMAN. Psychological and sociological. HENRY (364) MAUDSLEY, M.D. Pp. vi+386. Macmillan & Co., London. Pr. 12s. net.

DR MAUDSLEY'S first book on the "Physiology and Pathology of Mind," published nearly fifty years ago, brought him into the very first rank of scientific thinkers on mental processes in health and disease. The present work is in keeping with the great reputation which he has made for himself, and is a contribution of great value to the principle of the essential unity of the human organism and its continuity with the rest of nature's processes.

The book is divided into two parts, the first psychological and sociological, the second psychological and social. The fundamental point is that a true psychology must be founded on a clear recog-

nition of the essential continuity of nature and "the constant flux of things in which all so-called causes are effects, and all so-called effects causes." "Mind is to be conceived as a part of the nature which its organisation is in fact." "Psychology must come down from its misty heights and abstractions to the positive study of biology and lay its foundations solidly in physical science if it is to be of any value." "The whole body enters into the contribution of every mood, thought, and feeling." Man's self-conscious personality is "the emergent conscious quintessence of the registered experiences of the underlying bodily self." "To deride the notion of mechanism as anywise applicable to life, and life in mind, is the natural error of him who, ignorant of the exquisitely subtle physical and chemical processes at work in the simplest vital substance, thinks grossly only of inert matter and its mechanism as exemplified in the structure of a wheel-barrow or a steam engine." The whole progress of the race is conditioned by two strongly persistent strains which belong to the lowest as well as the highest forms of life—the instinct of reproduction and the instinct of self-preservation. "Life is in essence self-seeking and self-assertive in all its forms." The development of the human species rests at bottom in its animal nature in the vital struggle. "Materialism neither can nor ought to be got rid of." "Individuality is always a connected part of the whole, infused by it, and no more vitally detached from it than the organ from the body, the bud from the branch, or the whole plant from external nature. To separate essentially the components of mental life in brain as matter and the mind, and, having made the absolute separation, to declare mental function to be the independent function of the latter, is a gratuitously superfluous metaphysical assumption directly opposed to scientific observation which would, if true, render a positive mental science impossible."

Dr Maudsley expresses here what is obviously the culmination of the meditation and cogitation of a life-time. He gives an unusually clear exposition of physiological as opposed to vitalistic psychology, which presumes the existence of some inscrutable and unverifiable force apart from natural agencies. He considers it a delusion and conceit on the part of man to fancy himself the end of creation and measure of the universe, the final purpose for which the whole of reality has come into being. He thinks there is no evidence for this assumption, nor indeed any proof of purpose in creation at all. The sun is the probable source of life, and when it dies out man will cease to be. We know nothing of mind apart from body. Mind and matter may be essentially one and the same, termed either the one thing or the other according to the person's fancy.

It is impossible to discuss a book such as this in the short space devoted to a review; most of what it contains is highly controversial. It is treated here by a master mind, which is expressing its own views with a clarity and firmness of thought unfortunately rare. It is written from the viewpoint of a man widely read in philosophy as well as in the physiology and pathology of mind, and as such is worthy of the closest attention. The unbiased attitude of the writer will excite keen criticism from many quarters, and give the thinking reader much subject for thought.

INSANITY IN EVERY-DAY PRACTICE. E. G. YOUNGER, M.D. (365) Fourth Edition. Pp. x+134. Baillière, Tindall, & Cox. 1917. Pr. 4s. 6d. net.

THIS small book has been revised, and a new section added on neurasthenia, and another on psycho-analysis. It is essentially a book for the general practitioner, and is intended specially for those who are brought into touch with mental disease without having had any previous knowledge of insanity. It supplies simply the information required, and can accordingly be recommended.

MALINGERING, or the simulation of disease. A. BASSETT JONES, M.B., (366) and LLEWELLYN J. LLEWELLYN, M.B.; **with a chapter on malingering in relation to the eye**, by W. M. BEAUMONT. Pp. xxiii+708, with 5 plates. Wm. Heinemann, London. Pr. 25s. net.

THE authors of this monumental volume are already known by their previous book on fibrositis (*v. Review*, 1916, xiv., p. 192), and the same extensive clinical experience and analysis of detail has again been introduced into the present work.

They suggest that the study of malingering has been somewhat neglected by the scientific physician, who, more bent on establishing the features of true disease, has instinctively recoiled from the study of feigned disorders. In view of recent legislative innovations, with the increased opportunities they afford to the fraudulent-minded for deception, the need for systematising our knowledge regarding malingering, of perfecting our powers of discriminating between feigned and genuine disease, has become a matter of national importance. This can only be attained by keen realisation on our part of our diagnostic limitations, of the need for further research along the most scientific lines, correlated with a more lively sense of our obligations to the State, which looks primarily to the medical profession for its defence from this kind of imposition. To understand the feigned disorders, not only

must the normal aspect of disease be known, but also its aberrant manifestations, and especially the differentiation of "functional" from simulated disease.

The book is divided into five sections, termed respectively (1) general considerations, (2) malingering in relation to the nervous system, (3) malingering in relation to internal diseases, (4) malingering in relation to injuries and external injuries, and (5) measures for restriction of malingering.

The first chapter contains an historical introduction to the subject, and discusses the question of etiology. It is recorded that in the French Army, as the discipline of a corps approaches perfection, *pari passu* instances of simulated disease become less and less frequent; and that the incidence of malingering varies with the astuteness of the military surgeon. Pure malingering, *i.e.*, malingering without any substratum of true disease, is rare, but other varieties are common. The so-called increase of malingering is more apparent than real, and is referable to more precise diagnosis. Kuhn, in discussing the alleged frequency of feigned insanity, states that these cases occur in inverse ratio to the psychiatric knowledge of the physician. Malingering is "a specific form of simulation—a perversion or degradation of the imitative faculty which savours of atavism or regression to type," and may be classified into three groups: (1) Pure malingering, (2) partial malingering or exaggeration, and (3) false imputation. Exaggeration, either conscious or unconscious, is the most common form of malingering.

The differential diagnosis of malingering is treated at great length, no less than fifteen chapters being devoted to malingering in relation to the nervous system, including a chapter on malingering in relation to sight. The final section on measures for restriction of malingering discusses the question of prophylactic, disciplinary, and educational means. Malingering is essentially anti-social, and it is necessary to go to the very root of the matter and eradicate the cause—not an easy thing to do.

The volume covers a wide range of medicine, and the description of the examination of the nervous system, the differential diagnosis between simulation of disease, psychogenic disease, and early organic disease, and the proper interpretation of symptoms, quite apart from other systems, is worthy of the greatest praise.

Two other points also stand out prominently—first, the wide and not unsympathetic knowledge of human nature shown; and second, the high literary merit, which is rare in works of this kind.

Review

of

Neurology and Psychiatry

Original Articles

OBSERVATIONS ON REFLEX PHENOMENA IN CASES OF SPINAL INJURY IN MAN.

By **T. GRAHAM BROWN** and **R. M. STEWART**
(From the Neurological Department, British Salonica Force).¹

I. INTRODUCTION.

THE so-called "defence-reflex" in man is but an instance of that flexion-reflex of the limbs which has been observed in all other limbed vertebrates hitherto investigated.

The following observations we believe to demonstrate in man phenomena which have been found in other mammals, but not yet shown for the human subject. Before we proceed to their description it will be useful to summarise the characters of the flexion-reflex. The flexion-reflex of a limb is part of the general reaction of the animal to stimulation, particularly of the skin of the same limb. It is accompanied, as part of the same reaction, by an extension of the opposite limb. The flexion-reflex is really an abstraction—a "type reflex" (Sherrington)—composed of all the innumerable flexion-reflexes which are evoked by stimulation of the innumerable sensory end-organs of the limb. Its receptive field embraces all these sense organs—not only those in the skin itself, but also those of the deeper structures—tendons, muscles,

¹ We are indebted to Lieut.-Colonel N. J. C. Rutherford, D.S.O., the Officer Commanding the general hospital in which this department is situated, for permission to publish this paper.

and joints. In it all the flexor muscles contract equally (Sherrington)—though with different degree in reflexes of different strengths. All the extensor muscles relax reciprocally. The reaction itself consists in two parts, the immediate reaction and the after-reaction. The immediate reaction is that of flexor contraction and extensor relaxation, and occurs during the continuation of the stimulus, although in weak responses all reaction may disappear while the stimulus still persists. The after-reaction is that which follows upon cessation of stimulation. It often consists in extensor contraction and flexor relaxation (the “extensor rebound” of Sherrington), but it may also appear as a sharp increment of flexion (Graham Brown), while sometimes alternate flexion and extension may be rhythmically repeated. Very weak stimuli normally give not flexion but extension in the same limb. It occasionally happens that the response in the opposite limb is one of flexion and not extension (Graham Brown and Sherrington). When stimuli are applied to both limbs simultaneously the reactions in either are an algebraic summation of the reactions which occur when either limb is stimulated separately (Sherrington). If one stimulus is made to cease before the other, the reaction in the remaining period of stimulation is an algebraic summation of the “rebound” effect from the stimulus which has ceased, and the immediate effect of the stimulus which still continues (Graham Brown). If the two stimuli are suitably graded the reaction during the period of double stimulation may consist in a rhythmic alternation of flexion and extension (Forbes, Sherrington, Graham Brown). It may be noted that not all parts of the receptive field are equally excitable. The skin of the sole of the foot seems peculiarly sensitive. It may also be noted that the reactions are obtained with greater ease and regularity in the spinal animal—that is, after a transverse lesion which cuts off the higher parts of the nervous system. One further point is of interest. A rapid division of the spinal cord about the level of the ninth or tenth thoracic segment in the decerebrate animal evokes rhythmic movements of progression in the lower limbs (Graham Brown). A final point of importance is this: that in the mammal the movements of the toes apparently do not act with quite the same regularity in the reflex as those at the other joints of the limb and that the toes and fingers enter but slightly into the state of decerebrate rigidity, *i.e.*, “reflex standing” (Sherrington).

II. THE RECEPTIVE FIELD IN MAN.

In the normal man the flexion-reflex is difficult to obtain with regularity, and when obtained is a partial reaction. The well-known plantar reflex, in which the great toe flexes, the foot is inverted, and the tensor fasciæ femoris contracts, is obtained on stimulation of the sole of the foot. This reaction of flexion of the great toe is, however, in all probability not a part of the true flexion-reflex. But it seems to demonstrate that a certain small portion of the general receptive field of the flexion-reflex in normal man is regularly capable of effective stimulation under ordinary conditions.

This part of the receptive field is almost the only one stimulation of which gives a constant reaction in the normal subject. But certain stimuli applied to the back of the leg give a reaction which has not hitherto been described. If the leg be firmly grasped above the ankle, and the tendo Achillis be pressed against the posterior surface of the tibia, the great toe flexes at the interphalangeal joint. Save for passive extension of the ankle, no other movement accompanies this. A similar movement is obtainable on deep pressure applied in either of the fossæ between the tendo Achillis and the external and internal malleoli respectively. The reaction strikes the observer very distinctly as a passive and not a reflex one. Yet it is not conditioned by the passive extension of the ankle produced by pressing upon the tendo Achillis—for if a similar passive extension is produced by pressing upwards against the posterior edge of the heel, the great toe extends slightly. It is possible that it is produced passively by pressure upon the tendon of flexor longus hallucis. But in this case it is difficult to explain why it occurs with regularity when deep pressure is applied in the fossa between the tendo Achillis and the external malleolus. The reaction reverses with complete reversal of the plantar reflex in cases of spinal lesion. It has been present in all normal individuals that we have examined.

In a few normal individuals the flexion reaction of the great toe is produced by deep pressure applied to the vastus internus and externus muscles, the gastrocnemius-soleus muscle, and when the skin on the postero-internal aspect of the leg is rubbed.

Summarising this, we may say that in the normal man the receptive field of stimulation which gives with regularity a reaction

is fragmentary. It consists in the sensory structures in (*a*) the skin of the sole of the foot; (*b*) vastus internus muscle and gastrocnemius-soleus; and (*c*) the deep structures above the ankle and posterior to the bones of the leg. The flexion-reflex of the limb may of course be obtained by strong stimuli applied to almost any part of its surface in normal man, but not with regularity as a response to ordinary stimuli. That the reactions in (*b*) are produced by the stimulation of structures in the muscles is shown by the fact that they are not obtained by a similar pressure applied to a fold of loose skin above the muscle.

In cases of spinal lesion the flexion-reflex of the limb may be evoked with regularity by stimuli applied to a wide receptive field. The response of the great toe is usually (but not always) one of extension. At the other joints of the limb flexion occurs. Because it has not been recognised that the receptive field for this reflex includes almost every sensory end-organ in the limb, the reaction as evoked from different receptive end-organs has unfortunately been given different names and described as specifically different reflexes. Thus we have "Babinski's reflex"—the flexion-reflex of the limb as evoked by stimuli applied to the skin of the sole of the foot; "Oppenheim's reflex"—as evoked by a vigorous friction of the skin along the postero-internal aspect of the leg; "Schäfer's reflex"—as evoked by pinching the tendo Achillis; "Gordon's reflex"—as evoked by firm pressure between the heads of the gastrocnemius; the "Mendel-Bechterew reflex"—as evoked by tapping the bases of the third and fourth metatarsals; and so on. These, of course, are not separate reflexes, but only different manners of evoking one and the same reflex, and it seems unnecessary to dignify these methods with special names. As is well known, in cases especially of complete transverse lesion of the spinal cord, the flexion-reflex of the lower limb—the "defence reflex"—may be evoked by a sharp pinch applied to almost any area of the skin on the same side of the body below the level of the lesion.

We have evoked the flexion-reflex of the limb in cases of spinal lesion in the following different manners, which are additional to those already described: pressure applied in the fossæ between the tendo Achillis and the external and internal malleoli respectively; pressure upon the tendons of the long extensors of the toes; pressure upon the tendon of tibialis anticus;

pressure upon the body of *tibialis anticus*; pressure upon *vastus internus* and *vastus externus*; pressure upon *rectus femoris*; pressure upon *tensor fasciæ femoris*; pressure upon anterior abdominal wall about five centimetres internal to anterior superior iliac spine.

In all these cases the possibility that the reaction was evoked by pressure merely upon the skin and deep subcutaneous structures was excluded. These observations indicate merely that the receptive field includes not only the whole of the skin of the lower limb and lower part of the trunk, but also the deeper structures such as muscles and tendons—as in the lower animals.

While the stimulation of the superficial and deep (exteroceptive and proprioceptive) parts of the common receptive field usually gives similar reactions, this apparently is not always so—as the following observation demonstrates. In a case of syringomyelia we observed the extension response of the great toe when the deep structures were stimulated by pressing upon the heads of the *gastrocnemius* muscle, or by firmly rubbing along the posterior-internal border of the tibia. Yet superficial stimuli applied to the sole of the foot gave the flexion response of the great toe.

III. THE EFFECTIVE FIELD OF THE FLEXION-REFLEX.

The effective field of the flexion-reflex includes all or nearly all of the muscles of the limb. Those muscles to which the generic name “flexor” is applied, contract. The extensors relax (the terms flexor and extensor are physiological ones—thus *gastrocnemius* is physiologically an extensor, and *rectus femoris* is physiologically a flexor). In man it is difficult to demonstrate the phenomenon of muscular relaxation. We have observed the following muscles to contract in cases of transverse lesion of the spinal cord: extensors of toes, abductors of toes, flexors of the ankle, hamstrings, adductors of the thigh, *rectus femoris* (but not the other portions of quadriceps), *tensor fasciæ femoris*, the muscles of both sides of the anterior abdominal wall above and below the umbilicus. It is an interesting point that *rectus femoris* should contract but not the other parts of quadriceps. These latter are functionally extensors of the knee. *Rectus femoris* is a flexor of the hip, and has been shown experimentally in other mammals to be functionally distinct from the *vasti* and *quadratus*

femoris. The bilateral contraction of the muscles of the anterior abdominal wall is also a point of interest. As far as we know, it has not been observed in other mammals—probably because it has not been looked for. We have observed it to occur with regularity in a case of complete division of the spinal cord about the level of the fourth or fifth thoracic segment.

IV. THE REFLEX REACTION—IMMEDIATE REFLEX PHENOMENA.

In normal man the flexion-reflex of the limb is evoked with regularity only when strong stimuli are used, and is even then usually not a very complete reaction. In cases of lesion of the spinal cord a much more complete and regular reaction may be obtained if a sufficient time (three to eight weeks) has elapsed since the infliction of the injury. The phenomena of the reaction itself include those evoked by stimulation of one limb alone (simple reflex phenomena); and those evoked by stimulation of both limbs together (compound phenomena), which will be considered later.

The simple reaction consists in flexion at hip, knee, ankle, along with extension of hallux and sometimes abduction of the toes, all on the same side of the body. It is sometimes also accompanied by flexion of the lumbar spine (contraction of anterior abdominal muscles). These movements are all those of the stimulated limb, but they are accompanied by movements in the lower limb of the opposite side. These crossed movements may consist in extension of the limb at hip, knee, ankle (equivalent to the "crossed extension-reflex" of other mammals), or sometimes of flexion (equivalent to the less common "crossed flexion-reflex" of other mammals).

The "immediate" phenomena of the reflex are those which occur during the period of application of the stimulus (for instance, a sustained pinch of a fold of the skin). Throughout this period the flexion of the limb may be sustained; there is a short period of latency after the commencement of stimulation. Then the limb begins to flex, and the flexion gradually increases in value until a maximum is reached. It may then remain at this limit of flexion throughout the remainder of the period of stimulation, or the flexion may gradually relax—it may indeed entirely disappear

during stimulation if the stimulus is weak and sufficiently long sustained.

The reaction itself appears to be greater the stronger is the stimulus, but it is easily fatigued. In the reflex, flexion occurs in the effective field already described, and is accompanied by extension of the hallux and of the other toes, and often by abduction of the four outer toes ("fan movements"). A point of interest lies in the apparent dissociation of this movement of the toes from the flexion at ankle, knee, and hip. Thus in one case we obtained a well sustained flexion-reflex on maintained deep pressure upon the tibialis anticus muscle. The toe movements, however, appeared only at the commencement of stimulation and disappeared during the further continuance of the stimulus, although strong flexion persisted at ankle, knee, and hip. In some cases the flexion-reflex in the limb of the stimulated side not only dies away during stimulation, but is again re-established, although the stimulus itself is continued without alteration in value. The limb then performs rhythmic alternations of movement which will be described later.

A part of the immediate phenomena of the flexion-reflex are the movements which occur in the limb on the opposite side. In other mammals, under experimental conditions, the movement of the opposite limb is usually one of extension (the "crossed extension-reflex"), but is occasionally one of flexion. We have observed both these phenomena in man. The crossed extension-reflex occurs *pari passu* with the flexion-reflex of the limb of the same side. That is to say, that the movements of the two limbs are accurately reciprocal. The crossed extension-reflex is best seen where there is a certain amount of tonic flexion in the limb of the opposite side to that stimulated. Extension of the toes rarely accompanies the extension-reflex of the crossed limb, but is sometimes seen.

Occasionally we have observed the crossed reaction to be one of flexion. This crossed flexion-reflex seems to be favoured by the presence in the limb of a state of extension before the application of the stimulus. The crossed flexion-reflex may occur in response to stimuli so weak that no movement occurs in the stimulated limb. Its latency is long. Where the uncrossed flexion-reflex is transient and dies away, its disappearance phase (that is, the phase of uncrossed extension) may be accompanied by

flexion in the crossed limb. It is possible that the seeming pure flexion of the crossed limb, where no movement occurs in the uncrossed limb, is really a reaction of this nature—that is, that it is the accompaniment of the disappearance phase of an uncrossed flexion-reflex which could not be detected by the ordinary methods of observation. In this connection it may be remarked that in the case which exhibited this phenomena, flexion “rebound” phenomena (to be described later) were of greater extent than the immediate reflex phenomena.

These observations seem to show that, as in other mammals, the immediate phenomena of the reactions in the two limbs are accurately alternate. They seem to hint that the movements of the toes are by no means so stereotyped a part of the reflex as are the movements of flexion at ankle, knee, and hip. As we have said above, the extension movement of the toes does not necessarily proceed *pari passu* with the flexion movements at ankle, hip, and knee in the uncrossed reflex—yet the movements at these last-mentioned three joints seem to be fixed in relation the one to the other. Extension of the toes, as we have also observed above, may or may not accompany the crossed extension-reflex. In addition to these observations, we may note that a well maintained uncrossed flexion-reflex may be accompanied by phasic movements of the toes—that is, by rhythmically repeated extensions and flexions. The movements of the two lower limbs are not always, however, accurately alternate. Especially in response to pressure applied to the anterior abdominal wall opposite and near to the anterior superior iliac spine of one side, we have observed synchronous movements of flexion in the two lower limbs. These movements were markedly phasic—primary flexion giving place to extension during the continuation of the stimulus; but as far as we could judge, the movements of flexion in the two limbs were nearly equal in strength and absolutely synchronous in time, as were also the secondary movements of extension. We may remark here that although, in the ordinary reaction, a crossed extension movement accompanied an uncrossed flexion one, yet the adductors of the thigh often, if not invariably, contracted synchronously on the two sides. This movement is in itself one of some interest, for it may be connected with the adjustment of equilibrium necessary in the upright attitude, when one lower limb is raised from the ground. Thus the adduction of the extended limb is

part of a movement which tends to bring the centre of the body into the perpendicular line through the heel which remains upon the ground, while the adduction of the flexed limb also helps the movement by bringing the main mass of that limb towards the antero-posterior perpendicular plane which passes through that line.

V. "REFLEX REVERSAL" IN IMMEDIATE REFLEX PHENOMENA.

Although the rule of reflex reaction in the lower limbs of the mammal is that flexion occurs in the limb of the side stimulated, and extension in the crossed limb, under certain conditions the reactions may be made to reverse. Of these conditions one is the state of posture of the limbs at the time when the stimulus is applied. In a case of complete transverse division of the spinal cord we have observed this phenomenon of reversal in the movements of the crossed leg, as we have stated above. The uncrossed flexion-reflex of the right lower limb was accompanied by a well-marked extension-reflex in the crossed limb, if that limb was in a posture of maintained flexion when the stimulus was applied. If, however, that limb was in a state of extension, the crossed reaction was one of flexion. The respective movements of flexion and extension were observed to occur at ankle, knee, and hip. Another condition under which reflex reversal may appear is repetition of stimulation. We have seen this reversal in the case of the plantar reflex in a man suffering from compression of the cervical spinal cord by tumour, and we believe that this form of reversal of the plantar reflex is by no means rare. The reflex to a fresh stimulus was at first one of double phase. Well-marked flexion of hallux at the metacarpo-phalangeal joint (no movement at inter-phalangeal joint) was of short duration, and rapidly succeeded by well-marked extension, which was more maintained. On repetition of the stimulus, the reaction finally became one of pure extension of the hallux, the primary flexion phase disappearing. This reversal was seen on the left side only.

VI. "REBOUND" PHENOMENA (SUCCESSIVE REFLEX PHENOMENA).

In the mammal the final withdrawal of a sustained stimulus is followed by phenomena which are best termed "successive

reflex phenomena," in contradistinction to the "immediate" phenomena which occur during the period of stimulation. In the uncrossed limb the immediate flexion is succeeded by a phase of slow decrease of flexion—the so-called "flexion after-discharge." In strong reactions this is perhaps the usual successive phenomenon. Occasionally, however, on withdrawal of the stimulus, the uncrossed limb exhibits a sharp increase of the flexion then present in it—termed "flexor rebound after contraction." Yet again, on withdrawal of the stimulus, there may be a sharp relaxation of the flexion then in being—"flexor relaxation after contraction." It is obvious that the flexion after-discharge is only a slow form of flexor relaxation. In the crossed limb there are usually reciprocal movements, but often no movements occur at all. The most common successive phenomenon in the crossed limb is flexion. It should be noted that flexor relaxation is synonymous with extension, and that in the mammal it is accompanied by reciprocal contraction on the extensor muscles of the same limb.

In a case of complete section of the dorsal spinal cord in man the successive reflex phenomena were very distinct. In the uncrossed limb the successive phenomenon almost invariably took the form of an increase of flexion. This usually lasted for one or two seconds only, and having reached its maximum gave place to rapid relaxation. If the stimulus was sufficiently weak there might be no obvious movement of the limb during its period of application, but a well-marked flexion "rebound" on its withdrawal. This is a phenomenon frequently seen in other mammals. Stronger stimuli gave a transient flexion which disappeared during the period of stimulation, withdrawal of the stimulus being followed by a flexion rebound of yet greater extent. A still stronger stimulus conditioned flexion throughout its whole period, and an increase of flexion on its withdrawal. But if the period of stimulation was long continued, there was no flexion rebound, only a flexion "after-discharge." The rapidity of decrease of flexion in the after-discharge varied, and might be comparatively great.

As we have stated before, extension of the hallux and other toes accompanied the uncrossed flexion-reflex, but was more transient than the flexion at ankle, knee, and hip in strong reactions. It almost invariably disappeared during the period

of stimulation. It was again seen on withdrawal of the stimulus where flexion rebound occurred.

In the crossed limb reciprocal successive phenomena were evident. On withdrawal of the stimulus an increase in extension was observed to be nearly synchronous with the increase of flexion in the uncrossed flexion "rebound." This was, however, not always easy to observe, as it needs a background of original flexion for its proper exhibition. This extension "rebound" in the crossed limb was often followed by a movement of flexion in it. The flexion might be of very great extent, and indeed was often the only successive phenomenon in the crossed limb. This was especially the case where that limb was originally in a posture of extension, and it appeared to be exactly synchronous in its phase of increase with the phase of decrease of the flexion "rebound" in the uncrossed limb. In other words, the successive reflex reactions in the two limbs were reciprocally alternate in character.

Just as nearly synchronous contraction of the adductors of the two thighs was observed in the "immediate" phenomenon, so too was it observed in the successive phenomenon when it had died away before the termination of stimulation. But whereas the uncrossed adductors appeared to commence to contract shortly before the crossed ones in the "immediate" phenomenon (we estimated the difference in time at about 0.25 second), in the successive phenomenon the contraction appeared to be synchronous on the two sides. On one occasion flexion "rebound" appeared to be nearly synchronous in the two limbs. When there is flexion "after-discharge" (that is, slow decrease of flexion) in the uncrossed limb, there may be a reciprocal slow increase of flexion in the crossed limb. Phasic increase and decrease of extension of the toes was occasionally seen in the immediate reflex phenomenon of the uncrossed limb, especially when the stimulus was a sustained pinch of the skin. Similar phasic movements of the toes would then accompany a well-marked and sustained flexion rebound in that limb.

Stimuli applied to the lower part of the abdomen above or near Poupart's ligament seemed to be peculiar, in that nearly synchronous flexions occurred as immediate and successive reflex phenomena in both limbs. They were accompanied by extension of the toes of both sides. The movements in the two limbs

might be apparently of equal extent, and there was absolutely no difference observable in their times of commencement. Contraction of the crossed and uncrossed abdominal muscles was part of the successive phenomena. The immediate and successive contraction of the abdominal muscles was observed to include rectus abdominis and the obliques of both sides.

VII. COMPOUND REFLEX PHENOMENA.

In the mammal, when stimuli which evoke antagonistic movements in a limb are applied together, the resultant reaction is an algebraic summation of the two (Sherrington). Thus, if one stimulus gives a marked flexion and the other a slight extension, when both are applied together the limb gives a movement of flexion less than that seen when the flexion-producing stimulus is applied alone. We have observed this phenomenon in man. Thus when the stimuli used were such as to evoke slight flexion in either limb when given singly, little or no movement might be seen when they were applied together—the stimuli, of course, being given to both limbs simultaneously. If the stimuli were stronger, flexion was observed to occur in both limbs, but to be smaller in extent than when either stimulus was given alone. If a strong stimulus was applied to the right limb and a weak one to the left, flexion was observed in the former and extension in the latter, but both these movements were smaller than the movements obtained on applying the strong stimulus to the right limb alone. Slight flexion and not extension might occur in the weaker stimulated limb. Exactly similar algebraic summations were observed in the successive reflex phenomena. In the case of stimulation of the abdominal wall by deep pressure on one side near Poupart's ligament, the immediate reaction was one of synchronous flexion in the two limbs, but the flexion, though great in extent, was not well maintained. It gave place to extension during the continuation of the stimulus, and on withdrawal of the stimulus a similar movement to flexion, succeeded by extension, was observed in the two limbs. The duration of the flexion was about two seconds. When stimuli were applied on the two sides synchronously, the flexion movement in either limb was greater than that which occurred when

a single stimulus was given, but its duration did not appear to be markedly altered. This increase in flexion was seen in the immediate and successive phenomena.

VIII. RHYTHMIC PHENOMENA.

Rhythmic reflex phenomena are not uncommon in the mammal in response to an arrhythmic stimulus. They are best obtained when nearly equal antagonistic stimuli are matched against each other, as one of us has shown, but they may also be obtained in response to a maintained stimulus applied to a single limb. This is perhaps especially the case immediately after the division of the thoracic cord in its lower part (eighth to tenth segments). It is perhaps significant for what we have to say, that in the decerebrate cat rapid division of the spinal cord at this level often starts well-marked rhythmic movements of progression, which may persist for a minute or more in the lower limbs. With the exception of the scratch-reflex, the rhythmic reflexes of the lower limbs of the mammal are more or less incomplete forms of progression. We have observed these to occur in man. In a case of complete division of the spinal cord in the upper thoracic region rhythmic reflexes were hard to obtain. This case was one of a Bulgar, in whom the spinal cord was cut across by a bayonet in the fourth thoracic segment. Rhythmic reflexes were not obtained during the application of antagonistic stimuli to the two limbs, but on unilateral stimulation occasionally an indication of rhythmic rebound phenomena was obtained. Alternate flexions occurred in the two lower limbs, but rarely more than two flexion phases were seen in either limb. On pinching the skin of the right lower limb, rhythmic movements occurred in the toes of the same limb, both as immediate and successive phenomena, but the flexion at the other joints of the limb was not rhythmic. In this case automatic movements occasionally occurred alternately in the two limbs. The movement in either limb consisted in flexion followed by extension—the whole double phase lasting one or two seconds. These movements, however, were not again repeated in the limb.

In a Serbian who had sustained an almost complete division of the spinal cord at the seventh thoracic segment we found it possible to obtain rhythmic alternate movements of both lower limbs many times repeated by simultaneous stimulation of the

soles of both feet. In a case of a tumour compressing the lower cervical segments of the spinal cord, and probably affecting the upper part of the thoracic spinal cord (not yet confirmed), well-marked rhythmic movements were obtained. On maintained deep pressure between tendo Achillis and the left internal malleolus, well-marked, long-maintained rhythmic movements were observed, especially in hallux. The rate of the movements was twenty-five in thirty-five seconds. If a similar stimulus was then applied on the right side, the rhythmic movements disappeared. At first there was a transient increase of extension in the left hallux, followed by rapid decrease, and slow increase again. When the stimulus was removed from the right side (being retained on the left), the rhythmic movements reappeared in the left hallux. They also might break through the double stimulation if that was long maintained, and further increased when the right stimulus was then removed.

In a case of transverse myelitis, the prick of a pin on the sole of either foot started rhythmic alternate movements of flexion and extension at ankle, knee, and hip of both lower limbs. These were not of great extent, and were comparatively slow—the phase repeating in about two seconds. In this case, and on the evidence of those movements, we guessed the level of the lesion to be in the lower thoracic part of the spinal cord. The myelitis was actually at the sixth thoracic segment.

In a case of acute polyneuritis, rhythmic involuntary movements of the toes were observed on bilateral stimulation of the soles of the feet, and rhythmic alternate movements occurred in both lower limbs. In either limb these consisted in flexion at ankle, knee, and hip, followed by extension. For a short period the plantar reflexes were extension in type, and he had a difficulty in micturition, which later disappeared. He also had lumbar pains. In this case we suspected myelitis, and again guessed the level from these phenomena to be in the lower thoracic region of the spinal cord. A transverse myelitis was found in the eighth thoracic segment.

IX. ON THE DIAGNOSTIC VALUE OF RHYTHMIC REACTIONS.

We have spoken above of the presence of rhythmic alternate movements of the lower limbs in response to stimuli which are

applied either to one lower limb alone, or to both lower limbs simultaneously. These rhythmic movements are really forms of the so-called "stepping reflex" which is the basis of the movements of progression in the limbed vertebrates. When they occur in man the rhythmic movements are shallow—that is to say, they are of no great extent at any joint in the limb. They are extremely like the so-called "narcosis progression" described by one of us in other mammals. They are similar to the movements found by one of us to occur immediately after division of the spinal cord in the lower thoracic part in the decerebrate cat.

The point to which we wish to draw attention here is the site of the lesion in the cases in which we have observed it. That site was the sixth, seventh, or eighth thoracic spinal segment in the cases in which definite evidence was obtained.

It seems to us legitimate to emphasise the possibility that the occurrence of these rhythmic reactions in man may be of diagnostic value in pointing to the site of the lesion as in the lower thoracic spinal cord, and probably in the sixth to ninth segments. The occurrence of the phenomenon would seem also to point to a complete, or nearly complete, compression or division of the spinal cord in that region.

X. CONCLUSIONS.

In this paper we have given an account of reflex phenomena encountered in man. The greater part of these have been observed previously in other mammals under experimental conditions. Many of them are described here for the first time as they occur in man. This does not, however, mean that they are of rare occurrence in the human subject, but only that they have probably not been systematically sought before. A few of these observations would seem to be novel. One is perhaps of diagnostic significance. We think, however, that the phenomena which we have described are of chief interest in that they show that the limb reflexes of man are strictly comparable in their various characteristics with the limb reflexes of other vertebrates. Indeed, apart from the phenomena which can only be seen when the movements of isolated muscles are graphically recorded, almost all the characteristics and variations of the vertebrate limb reflex have been observed by us in man.

PERSISTENT CONGENITAL ŒDEMA OF THE LEGS (MILROY'S DISEASE) IN MOTHER AND DAUGHTER.

By J. D. ROLLESTON, M.D.

(With Plate 49.)

I RECENTLY had to examine a young married woman, aged 22, who offered herself as a daily worker in a military hospital. Apart from an attack of chorea at the age of 12, she said that she had always been strong and healthy, and her prepossessing appearance confirmed this. It was therefore somewhat of a shock, on examining the legs for varicose veins, to find that the left leg presented a deformity resembling elephantiasis (*v. figure*). From the knee downwards the limb showed a painless brawny œdema. Over the foot and ankle the condition was less obvious, because the œdema was reduced by the boot. The right leg showed a similar appearance, but in a very much less degree. No varicose veins were present in either leg. The maximum circumference measurements were:—

		Right Leg.	Left Leg.
Knee	-	12 inches	12 $\frac{3}{4}$ inches.
Calf	-	12 "	14 "
Instep	-	9 $\frac{1}{2}$ "	10 "

On hearing that her mother also had swollen legs I invited them both to come the following day, when I found that the mother, a woman aged 45, presented an exactly similar condition to that of her daughter, except that the right leg was more affected than the left. The maximum circumference measurements were:—

		Right Leg.	Left Leg.
Knee	-	13 inches	13 inches.
Calf	-	15 $\frac{3}{4}$ "	12 "
Instep	-	10 "	9 "

The mother informed me that her mother and her mother's sister were similarly affected. Her two other children, men aged 20 and 25 respectively, were normal as regards their legs, as was



also her daughter's only child, who died in infancy. She further stated that her œdema became less after a night's rest, and almost disappeared if for any reason she was compelled to keep to her bed for a week or more. In neither the mother nor the daughter was there any obvious cause for the œdema. In both the heart and lungs were normal, and the urine was free of albumin. In neither was there any local mechanical obstruction to the circulation, such as tight garters. Neither presented any other evidence of vasomotor disturbance, such as urticaria, cyanosis of the hands, or puffiness of the lids. Neither suffered much inconvenience from the œdema which had been present in both since birth, and had not been affected by pregnancy or menstruation. The only subjective symptom present was a feeling of heaviness in the affected limbs, especially after much walking. There was no history of acute exacerbations in either.

The two cases thus presented the four cardinal symptoms of the condition described by Milroy in 1892: (1) congenital character; (2) limitation of the œdema to the lower limbs; (3) persistence of the œdema; (4) entire absence of constitutional symptoms.

A special feature in both cases was the unilateral predominance of the œdema, the right leg in the mother, and the left leg in the daughter being chiefly affected.

As regards the occurrence of familial nervous disease to which Hope and French have called attention in connection with Milroy's disease, the daughter, as already stated, had had chorea in childhood, and one of her brothers was of a very nervous disposition.

Commenting on these cases at the Clinical Section of the Royal Society of Medicine, where they were shown on 11th May 1917, Dr F. Parkes Weber remarked that while non-familial examples of this form of segmental œdema were not uncommon, familial cases were extremely rare. He added that the same might be said of other diseases, such as in Recklinghausen's disease and multiple exostoses.

In addition to Milroy's original cases (nineteen cases in six generations), the only other examples of congenital and hereditary persistent œdema of the limbs which I can find in literature are those of Nonne (eight individuals affected with congenital elephantiasis in three generations) and Lortat-Jacob (congenital œdema of feet in three generations). Their pedigrees will be found in Dr William Bulloch's article on chronic hereditary trophœdema,

together with the pedigrees of other cases in which the œdema was hereditary but not congenital. As none of the recorded cases has come to autopsy, the pathogeny of the condition remains obscure. Various hypotheses have been put forward, such as congenital malformation of the trophic centres in the spinal cord for the cellular tissue (Meige), abnormal development of the mesoblast (Rapin), affection of the spinal centres for the lymph secretion (Valobra), insufficiency of the thyroid or thymus (Spiller), and endocrino-sympathetic dystrophy (Ayala).

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- (4) JOFSON, *Arch. of P.d.*, 1898, xv., p. 173.
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- (11) TOBIESEN, *Jahrb. f. Kinderheilk.*, 1899, xlix., p. 392.
- (12) VALOBRA, *Nouv. Icon. de la Salpêtrière*, 1905, xviii., p. 201.

Abstracts

ANATOMY.

THE ARTERIES OF THE PONS AND MEDULLA OBLONGATA— (367) PART III. J. S. B. STOPFORD, *Journ. of Anat.*, 1917, April.

THIS portion of the paper deals with the clinical application of Parts I. and II. (*v. Review*, 1916, xiv., p. 155, and 1916, xiv., p. 297). The symptoms to be expected from occlusion of the vessels of the hind brain at different levels are described and discussed. The wide variation in distribution of the arteries renders it inadvisable to associate emphatically a single group of symptoms with any one artery, and consequently it follows that a too stringent regard for the various syndromes must inevitably lead to errors in diagnosis.

The clinical significance to the neuro-vascular relationships in the skull is considered. Emphasis is placed particularly on the important relations between the optic nerve and chiasma, oculo-motor and abducent nerves, and certain blood vessels. Post mortem, there is repeated evidence of pressure on the optic nerve being exerted by a diseased internal carotid artery, and yet clinically this possible factor in the production of optic atrophy is persistently disregarded.

Reference is made to the way in which the oculo-motor and abducent nerves may be strangled, by the posterior cerebral and anterior inferior cerebellar arteries respectively, in cases of cerebral tumour or other conditions which are accompanied by any considerable alteration in the position of the brain stem.

AUTHOR'S ABSTRACT.

"PROBOSCIS PORES" IN CRANIATE VERTEBRATES: A SUG- (368) GESTION CONCERNING THE PREMANDIBULAR SOMITES AND HYPOPHYSIS. EDWIN S. GOODRICH, *Quart. Journ. of Micr. Sci.*, 1917, lxii., Dec., p. 539 (18 figs.).

GOODRICH gives an account of the complex histological structure of the epithelium lining Hatschek's pit in amphioxus, and of the development of this pit, and of the pre-oral pit from the left anterior coelomic sac and an ectodermal ingrowth respectively. The pre-oral pit becomes the ciliated wheel-organ of the adult, which was shown by Johannes Müller to drive a current of water

and food particles into the mouth. The ciliated cells of Hatschek's pit are of mesodermal origin, but the rod-bearing cells appear to come from the ectoderm. "The evidence is strongly in favour of Bateson's comparison of the opening of Hatschek's pit with the proboscis pore of balanoglossus and the water-pore of echinoderms. All these pores were originally paired. The anterior coelomic sacs of amphioxus are homologous with the premandibular somites of craniates. As shown by Ostroumoff, Dohrn, and Salvi"—in reptiles and in torpedo and raja—"these somites form tubular outgrowths opening into, or fusing with, the hypophysis—a connection comparable with the 'proboscis' pores of enteropneusta, cephalodiscus, and echinodermata. The premandibular, proboscis, and water-pores are all of the nature of coelomostomes. It is concluded that the hypophysis of the craniata is represented in amphioxus by the wheel-organ situated in front of the true mouth, and that its original function was probably to drive food into the alimentary canal." (It is interesting to note that Goodrich finds that in the three-day embryo of the duck the premandibular somites are intimately connected with the hypophysis.)

LEONARD J. KIDD.

PHYSIOLOGY.

THE RÔLE OF PRESSURE IN THE PHYSIOLOGICAL ACTION
 (369) **OF THE CEREBRO-SPINAL FLUID. ITS RELATIONS**
WITH THE FUNCTIONS OF THE CHOROID PLEXUSES
AND OF THE PINEAL GLAND. (*Le rôle de la pression dans*
l'action physiologique du liquide céphalo-rachidien. Ses rapports
avec les fonctions des plexus choroïdes et de la glande pinéale.)
 L. BARD, *Journ. de Physiol. et. d. Path. général*, 1917, xvii., Sept., p. 171.

BARD thinks that the regulation of the pressure of the cerebro-spinal fluid depends on two different mechanisms, one for slow and durable changes, the other for sudden modifications, for rapid transitory changes such as those which result from movements and attitudes of the head and trunk. The first depends on changes in the quantity of the fluid, of glandular nature (choroid plexus), the second on simple displacements, of vascular nature. The sudden fall of pressure which one can sometimes demonstrate in the course of lumbar puncture, in the case of syncope, shows the rôle which the circulation plays in the maintenance of the pressure of the cerebro-spinal fluid; this fall can hardly be explained otherwise than by rapid enfeeblement of the choroid

plexuses in connection with the arrest of the arterial impulse. "The richness of the choroid plexuses in blood vessels must render them very sensitive to vasomotor influences, and enable them to swell or diminish like all erectile tissues: doubtless it is by this mechanism that there is a sudden fall of pressure of the cerebro-spinal fluid in syncope; and by the same mechanism is explained the rapid regulation of pressure needed in changes of body attitudes. Vascular turgescence increases the volume of the ventricular contents, and therefore its tension and pressure against the ventricular walls; vascular diminution has a contrary action." In ascending the animal scale we see that the tufts of the choroid plexuses increase in complexity; Bard finds that in this respect the dog comes between ox and man. The great complexity in man is, he thinks, connected partly with the great development of his cerebral functions, and partly with the multiplicity and flexibility of his head movements, and with the rapid regulations of pressure demanded by his erect position.

"The absence of nervous papillæ in the choroid plexuses forces us to look elsewhere for the receptive organ of baroscopic impressions." Bard looks on the pineal body as the barometric perceptor and regulator of the pressure of the cerebro-spinal fluid. He has been struck by the great abundance of elastic fibres in the human pineal body, arranged in plexuses interlacing in all directions. (He makes the erroneous statement that the structure of the pineal has not been minutely studied!) "As the organ is ovoid, encapsulated, and precisely limited, the presence of these elastic fibres can have but one object, namely, to enable it to offer an elastic resistance to the pressures it is submitted to, and to enable it to resume spontaneously its form when the intensity of these pressures ceases or diminishes." Bard says that although the details are insufficiently precise we may conclude that the normal pressure of the cerebro-spinal fluid is an essential factor, probably even the predominant factor, of the physiological rôle of the cerebro-spinal fluid. This pressure has a certain fixity which the choroid plexus has to make stable and regularise by automatic mechanisms destined to compensate for the conditions, both physiological and pathological, which can modify it. "The pineal gland appears to be the organ for the regulation of the pressure of the cerebro-spinal fluid; it acts as a peripheral sensorial organ for baroscopic perception, and as a centre of automatism of this special function." (From Bard's description it is difficult to understand the nervous mechanism by which he supposes that the pineal body regulates the secretory function of the choroid plexus.)

LEONARD J. KIDD.

PHYSIOLOGICAL RESEARCHES ON THE SEMI-LUNAR GANGLIA.(370) **(Recherches physiologiques sur les ganglions semi-lunaires.)**ÉDOUARD RETIF, *Compt. rend. Soc. de Biol.*, 1918, lxxxi., 26 Jan., p. 82.

THE chief results of the writer's experiments on the semi-lunar ganglia of rabbits and dogs are as follows:—

1. Removal of the left semi-lunar ganglion of rabbits gives merely temporary diarrhœa and slight wasting.
2. Bilateral removal of the semi-lunar ganglia, or removal of the right ganglion, always has given symptoms followed by death in hypothermia.
3. This post-operative terminal hypothermia is connected with hypoglycæmia.
4. Extirpation of the semi-lunar ganglia gives a great loss of body weight.
5. All the operated animals have had diarrhœa, usually intermittent, and sometimes appearing but slowly.

The presence of this diarrhœa and the constant great wasting leads Retif to think that solarectomized animals are in a state of dyshydration.

LEONARD J. KIDD.

THE RECEPTION OF MECHANICAL STIMULI BY THE SKIN, (371) LATERAL-LINE ORGANS, AND EARS IN FISHES, ESPECIALLY IN AMIURUS.GEORGE HOWARD PARKER and ANNE P. VAN HEUSEN, *Amer. Journ. Physiol.*, 1917, xlv., Nov., p. 463 (1 fig., 4 tables).

As amiurus is active chiefly in the dark, the writers blindfolded their fishes by kid goggles stitched to the skin, and covering the eyes. A careful and elaborate technique was used. Conclusions:—

1. The skin of amiurus is stimulated by the dropping of water, by water currents, by a slow vibratory movement of the whole body of water, by the impact of a leaden ball on the slate wall of an aquarium, by the lower tones from a submerged telephone (43, 86, and 172 complete vibrations per second), but not by the higher tones (344, 688, 1,376, and 2,752 complete vibrations per second), nor by a whistle blown in the air. The responses to these stimuli are locomotor.

2. The lateral-line organs of amiurus are stimulated by a slow vibratory movement of the whole body of water, by the impact of a leaden ball on the slate wall of an aquarium, by the lower tones from a submerged telephone (43, 86, 172, and 344 vibrations), but not by the dropping of water, by water currents, by the higher tones from the telephone (688, 1,376, and 2,752 vibrations), nor

by a whistle blown in the air. The responses to these stimuli are inhibitions of those initiated through the skin and the ear.

3. The ear of *Amiurus* is stimulated by a slow vibratory movement of the whole body of water (utricle?), by the impact of a leaden ball on the slate wall of an aquarium (sacculus?), by certain tones from a submerged telephone (by 43, 86, 172, and 688, but not by 1,376 and 2,752 vibrations), by a whistle blown in the air, but not by the dropping of water, nor by water currents. The responses to these stimuli are locomotor. LEONARD J. KIDD.

ON THE GENESIS AND INHIBITION OF EXTENSOR RIGIDITY

(372) STANLEY COBB, ALBERT A. BAILEY, and PAUL R. HOLTZ, *Amer. Journ. Physiol.*, 1917, xlv., Sept., p. 239.

THE writers have in this experimental study tried to find out the exact centres involved in the genesis and inhibition of decerebrate rigidity: they use the word "genesis" as meaning "the main reflex centre," the ultimate source of the rigidity being the proprioceptive receptors. Conclusions:—

1. Stimulation of the superior cerebellar arm in decerebrate cats causes ipsilateral inhibition of their extensor rigidity.

2. The inhibition of decerebrate rigidity by stimulation of the cortex of the anterior lobe of the cerebellum (Sherrington, Thiele, Weed) appears to depend on stimulation of the underlying superior cerebellar arm.

3. A kind of extensor rigidity (not the typical "decerebrate rigidity" of Sherrington) is found in preparations transected below the red nucleus. This extensor tonus is lost when the transection injures the vestibular nuclei.

4. Stimulation of the superior cerebellar arm elicits no inhibition of extensor tonus when the red nuclei are not left in the preparation or when the dentate-rubral path is cut.

LEONARD J. KIDD.

EXPERIMENTAL RESEARCHES ON THE HYPOPHYSIS OF THE

(373) FROG, *LEPTODACTYLUS OCELLATUS* (L.) GIR. (*Recherches expérimentales sur l'hypophyse de la grenouille *Leptodactylus ocellatus* (L.) Gir.*) B. A. HOUSSAY, *Journ. de Physiol. et de Path. gen.*, 1917, xvii., Dec., p. 406 (5 figs.).

EXPERIMENTS on a common South American frog by means of hypophysectomy by Caselli's transparabasal or buccal route: the

effects of extracts of the frog's hypophysis were also studied.
Conclusions:—

1. The hypophysis of *Leptodactylus ocellatus* is composed of three parts, a glandular, an intermediate, and a nervous.
2. The glandular part contains two distinct types of cells—
(1) chromophilic, with acidophile and osmophile granules, and
(2) non-chromophilic.
3. The frog's hypophysis is not an organ essential to life.
4. Its total ablation frequently leads to early death, apparently independently of operative trauma. But prolonged survival of the animals for an indefinite time can be obtained sometimes.
5. After a dummy operation, the hypophysis being left *in situ*, the animals nearly always survive.
6. Preliminary graft of the hypophysis appears to prolong the survival of hypophysectomized frogs.
7. Hypophysectomy does not diminish the reflex excitability of the vagus centres.
8. The frog's hypophysial extract contains the substances which increase the arterial pressure and reinforce the cardiac systolic energy; it is galactagogue; it increases the contractions of the isolated oesophagus of the toad, dilates the renal vessels, and produces diuresis. These substances are therefore analogous to those which the hypophysis of other vertebrates contains.

LEONARD J. KIDD.

PSYCHOLOGY.

THE EMPLOYMENT OF THE CALCULUS OF PROBABILITY IN
(371) **PSYCHOLOGY.** (L'Emploi du calcul des probabilités en psychologie.) CH. JÉQUIER, *Arch. de Psychol.*, June 1917, vol. xvi., p. 197

A KNOWLEDGE of the theory of probability and of the mode of calculating probability is becoming of more and more importance in all the sciences. The aim of this article is to familiarise psychologists with this region of mathematics, and to estimate the value of the calculation of probability in the field of experimental psychology. There are fourteen figures.

MARGARET DRUMMOND.

PATHOLOGY.

A REVIEW OF THE NATURE AND FUNCTION OF THE
(375) **NEUROGLIA.** CHARLES RICKSHER, *Amer. Journ. Insan.*, 1917, July,
No. 1, p. 15.

A SHORT review of the work of Golgi, Cajal, Weigert, Marinesco,
Alzheimer, and others. D. K. HENDERSON.

FURTHER OBSERVATIONS ON EXPERIMENTAL TOXI-INFEC-
(376) **TION OF THE CENTRAL NERVOUS SYSTEM.** DAVID ORR
and Major Rows, *Brain*, 1917, xl.

THIS communication is a continuation of experimental work on the action of bacterial poisons upon the nervous system. Celloidin capsules containing a culture of *Staphylococcus aureus* were placed in contact with the common carotid artery in the neck, and two types of lesion were found to occur in the brain—(1) coagulation necrosis of the nerve cells in the cornu ammonis, in the cerebral cortex, and in the amygdaloid nucleus; (2) softening in the stratum moleculare of the cornu ammonis.

The nerve cells affected showed the deep diffuse coloration and shrinkage characteristic of necrosis. In the ischaemic softening the products of inflammation could be separated into four layers: (a) a central zone of degenerated red blood corpuscles and of degenerate nuclei; (b) outside this a zone of small round cells mixed with a few granular epithelioid cells; (c) a third and dense layer of epithelioid cells; and (d) a marginal zone of small round cells and proliferated neuroglia; here the adventitial cells of the vessels showed marked proliferation. Every stage of hyaline thrombosis of the vessels was observed.

It is important to note that the above areas alone are affected, and that they are supplied by the pial blood vessels.

The histological difference between the cortical and subcortical lesions suggests some difference in the vascular arrangement subserving the grey and adjacent white matter respectively. Both are supplied by pial vessels. Blockage of those supplying the white matter results in a softening; while the same lesion in the cortex causes coagulation necrosis of the nerve cells only. It is probable that the richness of the vascular network in the grey matter counterbalances the effects of mechanical interference with the vascular supply; at least this seems the most reasonable view to adopt while the whole question of the cerebral circulation is still so controversial.

The results of these experiments throw some light upon the genesis of the infantile cerebropathies, many varieties of which are now recognised to be of infective and inflammatory origin, and show how toxic hyaline thrombosis of capillaries and larger vessels can contribute to the production of nervous lesions of varying degree.

AUTHOR'S ABSTRACT.

CLINICAL NEUROLOGY.

ON THE INVESTIGATION OF THE ANKLE JERK. (*Da pesquisa (377) do reflexo achilleo.*) O. AYRES, *Brazil-Medico*, 1917, **xxi.**, p. 43.

AYRES recommends what he calls the "Brazilian method" for obtaining the ankle jerk in contradistinction to the positions recommended by Dejerine and Babinski. The patient lies on his belly, with one of his legs raised to a right angle. The doctor's left hand rests on the patient's sole, while he percusses the tendon Achillis with the hammer held in his right. A sudden rise of the foot indicates the presence of the reflex, while if the foot remains motionless, or almost motionless, the reflex either does not exist or is diminished.

The "Brazilian method" has the following advantages:—

1. The position is very convenient both for the patient and doctor.
2. The reflex can easily be obtained in paraplegia of the lower limbs, even when there is slight flexion contracture.
3. The personal coefficient of sight is eliminated.
4. The method is specially of value in comatose states and in children.

J. D. ROLLESTON.

A CASE OF CERVICAL RIB SIMULATING CERVICAL POTTS DISEASE. (*Un cas de côte cervicale supplémentaire simulant le mal de Pott cervical.*) JULES RENAULT and Mdlle. ROMME, *Arch. de Méd. des Enfants*, 1918, **xxi.**, Févr., p. 83 (2 figs.).

A GIRL, aged 13, was brought to hospital for anæmia. The position of her head strongly suggested cervical Pott's disease: her mother said that all the doctors of the city who had seen her had made this diagnosis. But careful clinical examination showed that the immobilisation of the flexed head was only apparent: all head movements were free, though rotation was very slightly limited, but equally on the two sides. There was no pain on

spinal percussion, and careful examination of the neck led to the diagnosis of cervical rib, which was confirmed by radioscopy. There were several other developmental abnormalities present, but there was nothing in the family history to explain their presence. The writers point out how such an erroneous diagnosis as Pott's disease in a case of cervical rib must lead to regrettable errors in both prognosis and treatment. LEONARD J. KIDD.

EARLY SYPHILITIC FATAL NON-SUPPURATIVE MENINGO-
(379) **ENCEPHALO-MYELITIS.** F. PARKES WEBER, *St Barts. Hosp. Reports*, 1916, I., pt. ii., p. 157.

A MAN, aged 32, who had been treated for secondary syphilis with neo-salvarsan and mercury a few months previously, was admitted to hospital with severe cerebro-spinal disease. Wassermann's reaction in the blood was negative, though positive in his wife. Death took place eleven days after admission. At the necropsy there was no definite macroscopic evidence of meningitis of the brain or cord, but microscopical examination showed very marked lymphocytic infiltration round the small blood vessels in the pia mater and in the spinal cord and cerebral cortex.

J. D. ROLLESTON.

CONTRIBUTION TO THE STUDY OF ANTERIOR SACRAL
(380) **MENINGOCELE.** (*Contribution à l'étude de la méninocéle sacrée antérieure.*) SUZANNE ROUX, *Rev. Méd. de la Suisse Romande*, 1918, xxxviii., Janv., p. 47 (3 figs.).

WHILE posterior sacral meningocele is relatively common, the anterior form is rare. The authoress finds that, if the non-viable cases be excluded, only twelve cases have been recorded during the last sixty years: all have been females, and pre-operative diagnosis has seldom been made. She reviews these cases and records a fresh one which Professor Roux operated on in 1914. The patient was a boy of 8 years who was brought to Professor Roux for incontinence of urine and obstinate constipation. A series of operations did much good. As to diagnosis, one should think of anterior sacral meningocele in the presence of an elastic or fluctuating tumour, of smooth surface, immobile, situated behind rectum or uterus, and joined to the sacrum, provided it be not directly connected with the uterine adnexa. Its general symptoms are almost the same as those of tumours of the small pelvis affecting rectum and bladder. As a rule it grows slowly, but after trauma or pregnancy may appear to develop very quickly. There is

usually obstinate constipation, disturbances of micturition, and pains in the lower extremities. Often other developmental anomalies are present; but they may be absent. As means of diagnosis we have puncture and radiography. While prognosis is sometimes favourable, this will depend largely on the severity of the operation needed. The authoress, in her conclusions, shows that a pre-operative diagnosis of anterior sacral meningocele is possible; the condition appears to be rare; the prognosis is relatively favourable; trauma or pregnancy may contribute to the evolution of a tumour or its discovery; the symptoms of compression of bladder and rectum come on rather slowly, her own patient being the only one who suffered from birth; nervous symptoms are rare before operation; concomitant malformations are often absent; and her own case is the only one of the male sex.

LEONARD J. KIDD.

EXTRA-MEDULLARY SPINAL CORD TUMOUR. CHARLES A. (381) ELSBERG, *New York Surg. Soc.*, 1917, April 11; *Annals of Surgery*, 1917, lxi., Oct., p. 510.

A WOMAN, aged 48, had signs of extra-medullary compression of the cord at the sixth thoracic level. Two years previously attacks of pain over left hip; soon afterwards difficulty in walking, both limbs becoming stiff, weak, and numb. For three and a half months complete paraplegia, with incontinence of urine and faeces. On examination, sensory and motor signs up to sixth thoracic segment. An extra-medullary tumour, the size of an almond, was removed from the posterior surface of the cord. It was a typical endothelioma, and very hard in consistency. Convalescence from operation was uneventful; early return of power; in four weeks regained control of bladder and rectum. Five months after operation she was quite well. Dr Elsberg stated that the small hard growths are much more apt to cause early irremediable damage to the cord than the larger and softer ones. In his experience the larger the tumour that was removed, the less the injury to the cord. He had seen a number of patients with small, easily removable growths in whom the paraplegia had persisted in spite of the removal of the growth.

LEONARD J. KIDD.

EXTRA-MEDULLARY SPINAL CORD TUMOUR. CHARLES A. (382) ELSBERG, *Trans. New York Surg. Soc.*, 1917, April 11; *Annals of Surgery*, 1917, lxi., Oct., p. 508.

DR ELSBERG showed a man in whom he had removed a spinal cord tumour from the level of the twelfth thoracic segment in

December 1916. There were all the symptoms of a growth at this level, but operation was much delayed on account of a history of lues and positive Wassermann. The tumour removed was a typical glioma, and recovery from operation was very satisfactory. Elsberg has several times met with a combination of syphilis and spinal cord tumour. He urges that if, with positive Wassermann, the symptoms of cord compression are not quickly improved by specific treatment, operation should be performed without delay. Spinal cord gummata usually improve very rapidly after the first salvarsan treatment. If symptoms do not rapidly improve, either the gumma is so large that its actual removal is indicated, or else the growth has nothing to do with the systemic syphilis. "It is wrong to permit a patient to become paraplegic while anti-specific treatment is tried. If treatment does not show immediate improvement, and if the spinal symptoms grow worse, the surgeon should not hesitate, but should proceed to operative treatment without delay."

LEONARD J. KIDD.

NOTE ON THE STAGES OF TABES DORSALIS—BASED ON THE (383) STUDY OF 240 CASES. M. GROSSMAN, *Med. Record*, 1917, xcii, p. 278.

THE writer's conclusions are as follows:—

1. The duration of the pre-ataxic period may be influenced by age.
2. The probable average pre-ataxic period is three years.
3. Women seem to have a shorter pre-ataxic period than men.
4. The average life expectancy of the bedridden tabetic is very much longer than that usually taught.
5. The average age of the immobilised tabetic is 53 years.
6. Most tabetics usually perpetuate the ataxic stage; in the small percentage of cases which become bedridden, owing to uncomplicated ataxia, the average duration of pre-ataxic period is 4.11 years.
7. Among those who become bedridden a short ataxic period usually follows a short pre-ataxic period.

J. D. ROLLESTON.

NEUROPATHIC AFFECTIONS OF JOINTS. LEO ELOESSER, *Annals (384) of Surgery*, 1917, lxvi, Aug., p. 201.

BONE and joint lesions corresponding to those found in tabes dorsalis may be induced experimentally in the limbs of cats by

severing the posterior nerve roots (sensory fibres) leading from the limb.

Severing of the posterior roots causes no atrophy of the bone.

Tabetic fractures and arthropathies have been produced in healthy animals, hence they cannot be ascribed primarily to lues or other infectious causes.

The course of a deforming arthritis is not characteristically altered by the addition of an analgesic factor, hence the cause of the Charcot joint is not to be sought in a simple deforming arthritis occurring in a tabetic.

Nothing in these experiments gives proof of the existence of trophic nerves.

Of three animals whose joints were subjected to operative trauma after having been previously rendered anæsthetic by resection of posterior roots, all rapidly developed Charcot lesions. Trauma in a limb rendered anæsthetic and analgesic experimentally leads to grotesque lesions of the bone and joints, which are in every way the counterparts of tabetic fractures and arthropathies; trauma and lack of the warning sense of pain are the cause of most tabetic bone and joint lesions.

A. NINIAN BRUCE.

ON MENINGEAL PHENOMENA DURING RECURRENT FEVER

(385) **IN CHILDREN.** (*Sur les phénomènes méningitiques pendant la fièvre récurrente chez les enfants.*) T. GANE and I. BUIA, *Compt. rend. Soc. de Biol.*, 1916, lxxix., p. 864.

DURING a recent epidemic of recurrent fever the writers met with definite meningeal symptoms without a leucocytic reaction in four cases, which should therefore be regarded as examples of meningism.

Two were brothers, aged 6 and 10 years; the third was a boy, aged 13; and the fourth a boy, aged 12 years.

J. D. ROLLESTON.

CYSTIC SEROUS MENINGITIS OF THE CRANIAL MENINGES.

(386) (*La meningite sierosa cistica delle meningi craniane.*) G. IZAR, *Riv. ital. di Neuropatol., Psichiatr. ed Elettroter.*, 1917, x., p. 29.

A PREVIOUSLY healthy woman, aged 24, developed the following symptoms: intense occipital headache, dysarthria, general weakness, diplopia, and inability to stand upright. On examination there were pain on percussion along the sagittal suture, slight nuchal rigidity, nystagmus, divergent strabismus of the right eye, paresis of the right lower facial and left hypoglossus, marked ataxia and

considerable weakness of the trunk muscles, especially on the right. Exploratory puncture showed a scanty collection of fluid above the cortex of the left frontal region. Complete recovery followed osteoplastic craniectomy. A review of the literature is appended.

J. D. ROLLESTON.

A CASE OF INFLUENZAL MENINGITIS. K. S. BHAT, *Lancet*, 1917, (387) ii., Sept. 8, p. 384 (illustr.).

A FATAL case in a girl, aged 14 months. The illness began with a sore throat, followed by "bronchitis" of about ten days' duration. Head retraction, neck rigidity, and moderate opisthotonus developed, with positive Kernig's sign, extensor plantar reflexes, and semi-consciousness. The case was at first thought to be one of tuberculous meningitis, but lumbar puncture gave issue to a slightly turbid fluid from which a "typical bacillus of the influenza type" was cultivated.

A. NINIAN BRUCE.

MENINGEAL REACTIONS IN SCARLET FEVER. (Réactions (388) méningées dans la scarlatine.) F. TRÉMOLIÈRES and L. CAUSSADE, *Bull. et mém. Soc. méd. Hôp. de Paris*, 1916, xl., p. 2129.

THE writers refer to the articles of Gouget and Benard (*v. Review*, 1909, vii., p. 198), Benard (*ibid.*, 1909, vii., p. 465), Cayrel and Weill (*ibid.*, 1911, ix., p. 680), and others, and record three personal cases of meningeal reaction at the onset of scarlet fever in young adults.

In the immense majority of cases of scarlet fever the cerebro-spinal fluid remains normal, but meningeal irritation may sometimes be manifested either by clinical signs or by changes in the cerebro-spinal fluid, or both.

1. In the simplest forms the meningeal irritation is revealed only by very slight chemical or cytological changes in the cerebro-spinal fluid, accompanied with ill-marked clinical symptoms. Possibly the scarlatinal neuralgia recently studied by Ramond and Schultz belongs to this group (*v. Review*, 1916, xiv., p. 238).

2. In other cases the meningeal symptoms are very definite, but there are no chemical or cytological changes in the cerebro-spinal fluid.

3. A more or less marked meningeal syndrome may be associated with a cellular reaction of varying intensity, *c.g.*, from 6-80 lymphocytes in a field.

4. The most intense reaction is shown by a meningeal syndrome

with a hæmorrhagic cerebro-spinal fluid, apart from any hæmorrhagic form of scarlet fever.

Meningeal reactions which are aseptic are more frequent than the microbial meningitis which occurs in scarlet fever as a complication of otitis or sinusitis, being found in four out of eighty-eight of the writers' cases.

The prognosis is not severe. The symptoms subside spontaneously or after lumbar puncture. J. D. ROLLESTON.

**CEREBRAL ABSCESS FOLLOWING OLD-STANDING MIDDLE-
(389) EAR DISEASE IN A MAN AGED 58 YEARS : OPERATION
AND RECOVERY.** G. VICTOR MILLER, *Clin. Journ.*, 1917, xlv.,
p. 52.

DEAFNESS in the right ear and discharge from it had persisted since boyhood. The symptoms were right hemiplegia and coma, preceded by pain in the ear and head. The patient was discharged cured twelve weeks after the operation. J. D. ROLLESTON.

**ABSCESS OF THE FRONTAL LOBE FOLLOWING ORBITAL
(390) CELLULITIS.** CHARLES A. ELSBERG, *Trans. New York Surg. Soc.*,
1917, April 11; *Annals of Surgery*, 1917, lxvi., Oct., p. 508.

A BOY of 13 was admitted in a much emaciated condition. Two months before an abscess of the right orbit had been opened: he recovered well from this operation. Three weeks later right-sided headaches, and then rapid loss of strength and flesh. Frequent vomiting attacks, and complete right ophthalmoplegia. On admission, very bad condition: pallor, emaciation, much neck rigidity, decided Kernig: had appearance of a case of tubercular meningitis. But he had double choked disc with slight left facial weakness. Brain abscess was discussed. Soon after admission had a convulsive attack affecting face and upper limbs, followed by slight weakness of left upper extremity. A few hours later another attack, after which the left upper extremity and left face were paralysed. Stupor in a few hours' time. Vertical incision was made in right fronto-parietal region: a button of bone removed with a trephine, the dura incised, and the brain aspirated. At a depth of 3 cm. below the cortex thick green pus was obtained. Packings soaked in iodine were placed all round the wound, and the exposed brain painted with iodine tincture. The lobe was then incised, and about 6 oz. of pus under marked pressure were evacuated. The frontal lobe was drained with two tubes. A very rapid convalescence: discharged entirely well, free from all

symptoms, two months after operation: eye-grounds normal, ophthalmoplegia gone.

Dr Elsberg stated that "there were some surgical principles in the treatment of brain abscess which were often neglected. A brain abscess should always be drained by two tubes, and these tubes should never be removed at the dressings, but should be gradually shortened. If, before an abscess of the brain is opened, packings of gauze soaked in tincture of iodine are placed under the dura all around the wound, there is practically no danger of meningeal infection. The drainage tubes should be held in place by sutures to the scalp, so that they can neither be pushed further into the brain nor become displaced in an outward direction. If these general surgical principles are followed, drainage of an abscess cavity in the brain can be carried out satisfactorily. It is wrong to remove a drainage tube from the brain and then attempt to reinsert it, because one can seldom get it into the same place again. In such attempts the neighbouring brain tissue will be sure to be injured."

LEONARD J. KIDD.

ACUTE GENERAL TUBERCULOSIS WITH LEFT OCULO-MOTOR

(391) **PARALYSIS AFTER MEASLES.** SIR JOHN MOORE, *Dublin Journ. Med. Sci.*, 1916, i., p. 174, and *Clin. Journ.*, 1916, xlv., p. 458.

THE patient was a girl, aged 4½ years, who had always been healthy until an attack of measles nine weeks previously. On the day before admission complete ptosis of the left eyelid developed. The evening temperature was 102° Fahr. On admission there was complete paralysis of the left third nerve. Death took place eleven days after admission. The autopsy showed disseminated tuberculosis of the lungs and abdominal organs, and extensive tuberculosis and basal meningitis. No involvement of the nucleus or of the intracranial course of either third nerve was observed on hardening and examining sections.

J. D. ROLLESTON.

A CASE OF MULTIPLE BRAIN SOFTENING. THOMAS WALMSLEY, (392) *Lancet*, 1917, ii., Sept. 8, p. 387 (2 figs.).

A WOMAN, aged 28, was admitted to Montrose Asylum on account of acute excitement of short duration with well-marked motor disturbance. She was discharged in eight months, but was re-admitted three years later, widowed, her husband having died in the interval in the same asylum from general paralysis. Her condition now rapidly progressed towards a complete dementia, interrupted by short periods of excitement, and terminating in a

stuporose state in which "she showed a distinct resemblance to the behaviour of a decerebrated frog."

Section of the cerebral hemispheres showed numerous cyst cavities, mostly confined to the white matter, absent from the cerebral cortex, and most numerous towards the anterior end of the brain, but extending through the mid-brain, cerebellum, pons, and medulla. The cysts appeared to have undergone a progressive increase in size, and the intervening brain tissue having undergone a pressure atrophy, neighbouring cysts had become confluent. These cysts had an aperture of communication with the surface of the brain. Microscopical examination showed the cyst wall was formed of brain tissue, there being no alteration in the density of the peripheral neuroglia, nor any appearance of a living membrane. There was no reactionary neuroglial overgrowth.

These lesions were thought to be due to multiple softenings, the result of multiple thrombosis of cardio-vascular genesis of the "end-artery" system of the brain. They were considered to be without relationship to any possible syphilitic infection.

A. NINIAN BRUCE.

CEREBRAL CYSTICERCOSIS. (*Contributo allo studio della cisti-
(393) cercosi cerebrale.*) G. BIONDI, *Riv. ital. di Neuropatol., Psichiat.
ed Elettrotel.*, 1917, x., pp. 10-21 and 51-69.

A RECORD of two cases with naked eye and micro-photographs.

Case 1.—A previously healthy man, aged 42, became subject to attacks of headache, which in the last weeks of life became continuous, with occasional exacerbations. Localised convulsions developed first in the limbs on the right side, often preceded by tonic spasms of the left facial, and followed by conjugate deviation of head and eyes to the right. Vertigo, choked discs, and later morning vomiting. Right hemianopsia, followed by complete blindness, hyperacusia, left hyposmia, and deviation of the tongue to the right. Diminution of muscular power with hypæsthesia in the right limbs, and hypæsthesia in the right half of the face. Abolition of the right cremasteric and abdominal reflexes. Severe psychical disturbances (torpor, somnolence, great diminution of capacity of fixation, loss of orientation, amnesia and paramnesia, hallucinations, &c.).

Death took place after an illness of two years' duration. The autopsy showed cysticercal meningitis of the base, internal hydrocephalus, and well-developed granular endymenitis.

Case 2.—The autopsy on an epileptic imbecile boy, aged 14, showed an obsolete cerebral cysticercosis. Histological examination

showed that the atypical development of the brain could be traced back to intra-uterine life, and was in no way concerned with the cysticercosis infection, though the latter probably aggravated the mental condition and tendency to epileptic attacks.

J. D. ROLLESTON.

CEREBRAL PALSY OF ONE UPPER LIMB. (*Paralysie cérébrale du* (394) *membre supérieur.*) VERGER, *Soc. de Méd. e. d. Chirurg. de Bordeaux*, 1918, 18 Janvier (*Jour. de Méd. de Bordeaux*, 1918, lxxxix., Fevr., p. 49).

A MAN, aged 61, in the course of a scuffle, was seized with a total palsy of his right arm, without pain or loss of consciousness: he did not know what part of his body was injured in the affray. This palsy rapidly improved; and, when Verger examined him, he found only a paresis of the right hand. All voluntary movements could be carried out, but only slowly and awkwardly. The radial reflex was exaggerated: slight contracture of fingers: sensibility to touch and prick preserved: astereognosis: normal electrical reactions: no muscular atrophy. "The regression of the paralytic phenomena, the exaggeration of the radial reflex, and the presence of astereognosis clearly agree with the idea of a monoplegia due to cortical softening." (The medico-legal aspect of such a case is touched upon.)

LEONARD J. KIDD.

CASE OF HEMIPLEGIA FOLLOWING PLEURAL EFFUSION. (395) G. DE B. TURTLE, *Lancet*, 1917, ii., Aug. 4, p. 161.

A MAN, aged 23, was admitted to hospital with pleural effusion, requiring to be several times aspirated. About a fortnight later he suddenly developed right hemiplegia, with complete motor aphasia, but without unconsciousness. The condition was attributed to an embolus being dislodged from a clot in the left pulmonary vein.

A. NINIAN BRUCE.

CRANIOTOMY FOR CEREBELLAR CYST. CHARLES A. ELSBERG, (396) *Trans. New York Surg. Soc.*, April 11, 1917; *Annals of Surgery*, 1917, lxvi., Oct., p. 507.

CASE of simple cyst of cerebellum in girl of 17. Admission January 1917. Eight months previously unsteadiness in walking, and attacks of headache and vomiting. Two months later diplopia and failing vision. On admission was bed-ridden, was extremely ataxic and unable to raise head from bed without vomiting.

Marked choked disc with signs of lesion of left lobe of cerebellum. Bilateral sub-occipital craniotomy on 13th January; left cerebellar lobe bulged very markedly on incision of dura, while there was no increase of tension on right side. Dark yellow fluid aspirated from left cerebellar lobe. About 5 c.c. evacuated; no evidence of a capsule. The wound in cerebellum was left wide open, and the soft parts closed in usual way. Uninterrupted convalescence; wound healed by primary union. Headache went at once; eye grounds normal in two weeks' time. Rapid diminution of all the ataxic symptoms. Patient discharged on 6th March, free from all symptoms.

LEONARD J. KIDD.

PROGRESSIVE MUSCULAR ATROPHY AND MULTIPLE
(397) **SCLEROSIS.** (*Atrofia muscolare progressiva e sclerosi multipla.*) G. BIONDI, *Riv. ital. di Neuropatol., Psichiatr. ed Elettrotet.*, 1917, x., p. 137.

A RECORD of the clinical symptoms and post-mortem findings of a case in which multiple sclerosis and progressive muscular atrophy developed simultaneously and independently in the same individual.

J. D. ROLLESTON.

NEUROFIBROMA IN THE CEREBELLO-PONTINE ANGLE.
(398) CHARLES A. ELSBERG, *Trans. New York Surg. Soc.*, 1917, April 11; *Annals of Surgery*, 1917, lxxvi., Oct., p. 509.

DR ELSBERG showed a man of 50 from whom he had removed a tumour from the cerebello-pontine angle fourteen months before. On admission there were the typical history, signs, and symptoms of tumour in right cerebello-pontine angle. In February 1916 bilateral suboccipital craniotomy: the tumour was enucleated inside of the capsule. Good recovery: symptoms gradually improving up to present time. The case was shown to call the attention of surgeons to a method of removal of these tumours which has been very satisfactory, and has markedly lessened the mortality from these operations. Small tumours (up to the size of a cherry) in the cerebello-pontine angle can be safely removed with their capsule. Large ones are usually closely adherent to the sides of pons and medulla: their removal *with* the capsule is very apt to cause secondary softening of these structures: a slight hæmorrhage here is only too apt to be fatal. Cerebello-pontine angle neurofibromas grow very slowly and are clinically benign: they harm only by pressure. It does no harm to leave the capsule behind. "Therefore if, when such a tumour is exposed, it is found to be of large size, no attempt should be made to remove it with

its capsule, but an incision should be made into the capsule, and with a sharp curette all the tumour should be scraped away. Although the capsule is usually fairly tough, care must be taken that it is not perforated. All the manipulations should be done with gentleness, and special care should be taken when curetting out the mesially placed portions of the growth. By this method all of the tumour is removed within its capsule, and the capsule is allowed to remain behind. The intra-capsular enucleation of tumours in the cerebello-pontine angle is a very satisfactory method of procedure, and the large majority of the patients recover from the operation."

LEONARD J. KIDD.

TENTACULAR GLIOSARCOMA OF CORPUS CALLOSUM. (Gliosarcome tentaculaire du corps calleux.) J. SARRAZÈS, *Gaz. Hebdomadaire des Sci. Méd. de Bordeaux*, 1918, xxxix., 24 Fév., p. 25.

A GLIOSARCOMA of the corpus callosum which extended, by tentacle-like expansions, forward into both cerebral hemispheres. A woman of 52 was admitted on 24th January 1913, and died four days later. For four years she had suffered from paroxysmal frontal headache, rather worse on the right side. Her intelligence seems to have been always very mediocre. Gait in 1909 was uncertain and titubating; obstinate constipation; boulimia; exaggerated thirst; gradually psychical disturbances developed in the shape of a discord between what she said and what she did. There was apraxia. She could not take care of herself. Increasing amnesia, verbal confusion, and disorientation in space. She then lapsed into chronic mental confusion, could not enter into a conversation, and gave erroneous and contradictory answers. Two years later (August 1911), when very feeble, physically and mentally, she had a stroke which left her paralysed in the lower limbs; the left lower limb, and then the left upper limb became contracted, the flexion of the fingers on the palm producing ulcerations. Sensibility to pin-prick was apparently blunted on both sides. She lay in right lateral decubitus, with her thigh and leg flexed but mobile, and not contracted on this side. She never complained, and was indifferent to everything. A month before admission in 1913 she spoke no more, and neither ate nor drank spontaneously. She passed her excreta in the bed. She had large bed sores on the buttocks, trochanters, thighs, heels, shoulders, and left neck, with fever, stiffness, and mental clouding. She could not be made to put out her tongue. Urine albuminous, with a skatol reaction, and slight excess of indican. There was flaccid paralysis. Her articular sensibility to limb-displacements was preserved, and was even increased on the left. The reflexes were lively, with Babinski

positive on left, doubtful on right. She succumbed to the infection of her multiple bed sores in a state of cardio-pulmonary collapse. Necropsy showed a gliosarcoma of the anterior region of the corpus callosum extending into both hemispheres. In the left hemisphere the germ of the callosum is invaded by the tumour, which at this level is of the size of a chestnut. It extends along the roof of the lateral ventricle, invades the lenticular nucleus, upper part of thalamus, and internal capsule; above, it fuses with the caudate nucleus, and extends into the centrum ovale: it almost reaches the cortex cerebri at the foot of the second frontal. The tumour is of flabby consistence, and has holes in it like those of Gruyère cheese: it shows no hæmorrhagic foci. It sends a polypiform prolongation into the lateral ventricle. From the left hemisphere the tumour passes into the right, infiltrating the septum lucidum, and involving the lenticular nucleus, internal capsule, and upper part of thalamus. There was degeneration of both pyramidal tracts. The paper ends with some remarks on the symptomatology of corpus callosum tumours, and the variability of the symptoms according to the part of the callosum affected. A lesion of the germ gives mental symptoms, with apraxia, disturbance of the association of ideas, and in the comprehension and proper use of words. There was in this case an absence of visual and fundus oculi changes, and preservation of pupillary reflexes.

LEONARD J. KIDD.

**NINE CASES OF HEREDITARY POLYDACTYLY IN FIVE
(400) GENERATIONS; POLYDACTYLY AND MENDELIANISM.**

(Neuf cas de polydactylie héréditaire au cours de cinq générations; la polydactylie dans ses rapports avec les lois de Mendel),
BENARD, *Nouv. Icon. de la Salpêtr.*, 1916-17, Nos. 2 and 3, p. 147.

THE documentary value of this communication is considerable. Full details are given of the occurrence of six fingers and six toes in a family in a country district of France. It may be said that "on the whole, and with certain reservations, the proportions indicated by Mendel are preserved, and that in this family polydactyly appears as a dominant."

S. A. K. WILSON.

**CLINICAL AND ANATOMICAL STUDY OF A CASE OF
(401) DUPUYTREN'S CONTRACTION.** (Un caso di malattia di

Dupuytren. Osservazione clinica e anatomica.) A. ZIVERI, *Riv. di Patol. nerv. e ment.*, 1917, xxii., p. 377.

THE patient was a man, aged 74, with Dupuytren's contraction in both hands, who died in an asylum to which he had been admitted

for manic-depressive insanity. No familial or hereditary history of Dupuytren's contraction could be obtained. Histological examination of the brain and cord showed thickening of the adventitia, and marked tortuosity of all the vessels and the presence of disintegrative lacunæ. The lesions were most marked in the cervical enlargement. Punctiform hæmorrhages were found in the upper part of the pons by the side of the aqueduct of Sylvius. There were no syringomyelic cavities.

J. D. ROLLESTON.

PARANOID TYPES IN SYPHILITIC DISEASE OF THE CENTRAL (402) NERVOUS SYSTEM. E. M. AUER, *Amer. Journ. Insan.*, 1917, July, No. 1, p. 53.

A CLINICAL report of six cases, in each of which a paranoid trend was the striking feature of the mental picture associated with positive serological findings.

D. K. HENDERSON.

THE NERVOUS SYMPTOMS OF POLYCYTHÆMIA VERA. (403) HENRY A. CHRISTIAN, *Amer. Journ. Med. Sci.*, 1917, cliv., Oct., p. 547.

OUT of ten cases observed by Christian, eight showed definite nervous symptoms, and in most of these the nervous disturbances were the chief cause of the patient's discomfort. These symptoms varied in duration from a few days to many years. The most frequent symptoms were headache and dizziness. Other common symptoms were visual disturbances, such as easily induced fatigue of eyes, blurring of vision, scotomata (often scintillating), transient blindness, hemianopia, and diplopia. Sensory disturbances, particularly paræsthesia, occurred. In several patients paresis and paralysis were seen. In one case these motor affections were transient, and recurred. In some cases the nervous symptoms were sufficiently focal to lead to diagnosis of brain tumour. In one case this feature was present over a period of ten years, and even after the blood count it was still thought possible that some of the focal symptoms were due to a cerebral tumour.

Conclusions.—Nervous symptoms are very frequent in polycythæmia vera, and usually they are the symptoms that lead the patient to seek medical advice. Since, quite often, polycythæmic patients fail to show cyanosis or erythema with these nervous symptoms, other cerebral lesions, such as brain tumour, are suspected. In the earlier stages such symptoms must result from simple circulatory disturbances; in the later stages cerebral softening or cerebral hæmorrhage and local vascular lesions, such as thrombosis, are often found.

LEONARD J. KIDD.

THE ARCH OF THE AORTA IN EXOPHTHALMIC GOITRE. (La crosse de l'aorte dans le goitre exophtalmique.) FOLLEY, *Compt. Rend. Acad. des Sci.*, 1918, clxvi., 18 Fév., p. 311.

THE writer finds that in all cases of exophthalmic goitre, both incomplete and the classical developed form, there is a dilatation of the aorta and its arch, as seen by radiography. This dilatation is a very early sign; it increases in degree as symptoms increase, and decreases as they decrease. He claims that these aortic signs should be regarded as proper symptoms of Basedow's disease; and that in very early cases, in the absence of any valvular signs, and even in what seem doubtful cases, the presence of aortic dilatation settles the diagnosis of that disease.

LEONARD J. KIDD.

IS THERE A HYPHEN IN THE NAME OF DR ARGYLL ROBERTSON? J. W. FARLOW, *Boston Med. and Surg. Journ.*, 1917, clxxvi., p. 147.

THE signature of an autograph letter from Dr Robertson in the Boston Medical Library clearly proves that there is no hyphen in his name.

J. D. ROLLESTON.

THE REACTION OF THE PUPIL TO COLOURED LIGHT. JAMES A. CUTTING, *Journ. of Nerv. and Ment. Dis.*, 1917, xlv., Oct., p. 246.

CONCLUSIONS:—

1. The pupil reacts differently to different coloured lights, giving a greater reaction in some than in others in the following order: white, yellow, reddish yellow, green, blue, and violet, thus following the luminosity of the spectral colours.

2. There is no reaction specific to different diseases—the same law holds good for the parietic as for the hysterical.

3. There is a distinct clinical value in using the green light as a "measure" for amplitude of reaction, which cannot be obtained from white light. It is a convenient method for measuring the amount of light necessary to produce a pupillary response. (As to the clinical value of the coloured lights Cutting writes:—"I have found a distinct advantage in the use of the eight-candle-power green light as a guide to the degree of sluggishness in a pupil. Particularly in testing the pupil in paresis was this true. Here a pupil was often found which would not react to an eight-candle-power green light, but would react to the ordinary white light. I find that it is much more satisfactory to say a pupil will

not respond to an eight-candle-power green light, than to say a pupil is somewhat sluggish to light." He also warns us to refrain from a diagnosis of paresis merely because both pupils fail to react to the green light; for alcoholics and also users of morphine and cocaine often give this sluggish response; but in unilateral sluggishness of response to green light we have a very significant sign. Very early in the case there may be a difference in the consensual reflex to coloured light.)

LEONARD J. KIDD.

BITEMPORAL HEMIOPIA: THE LATER STAGES AND THE
(407) **SPECIAL FEATURES OF THE SCOTOMA.** With an
examination of current theories of the mechanism of production
of the field defects. TRAQUAIR, *British Journal of Ophthalmology*,
1917, April, May, June, p. 216.

THE changes in the fields of vision in a typical case of bitemporal hemiopia begin in the upper outer quadrants of the fields, and then encroach upon the lower outer quadrants, so that the outer halves of the fields become lost from above downwards. This process may be more advanced in one field than in the other. With further progress the inner halves of the fields become defective from below upwards until the whole field is lost, the "later stages" of bitemporal hemiopia being those during which the nasal fields are affected.

Different views have been advanced as to whether vision remains last in the upper or in the lower nasal quadrants, and in the present paper this question is dealt with. An examination of published cases shows, if anything, a slight preponderance of evidence in favour of the up-in termination. No investigations, however, have hitherto been specially directed towards this point. In fourteen out of sixteen cases examined by the author, in which the field defects were sufficiently advanced, vision was found to be retained longest in the upper inner quadrants. Thus in typical cases of bitemporal hemiopia the defects begin in the upper outer quadrant of the field, and travel around the centre in a circular direction clockwise in the right field, and counter-clockwise in the left. The same circular mode of progress occurs in the scotoma which develops like a small field within the field. The central field changes are not necessarily contemporaneous with the peripheral defects.

The second part of the paper deals with the relation of the field defects to the various causal conditions, and the mechanism of production of the defects. The normal anatomical relations of the chiasma are discussed, and stress is laid upon the fact that

chiasma does not, as so often described, lie upon the optic groove of the sphenoid bone, but a considerable distance behind and above this position. The infundibulum lies in close contact with the lower surface of the posterior part of the chiasma, but the pituitary body is separated from the lower surface of the chiasma by a vertical distance of 5 to 10 mm.

The causes of the sixteen cases upon which the paper is based were: Acromegaly, four; maxillary antrum suppuration, one; infantilism with pituitary enlargement, one; pituitary tumour, four; cervical tuberculosis, one (probably associated with a tuberculous process in the neighbourhood of the chiasma); syphilis, two; and undiagnosed, three.

Scotomata were found to be present in the more active cases and absent in the more slowly progressive or stationary cases, such as chronic acromegaly. A ring scotoma which was present in one case of acromegaly is discussed in some detail. Eleven of the sixteen cases were X-rayed, and in seven sellar deformation, either deepening or flattening, was present; in four the sella was normal. The main feature elicited by the examination of the cases is the great variety of conditions, both general and local, which may be associated with the same type of field changes. In considering the mode of production of these changes it is therefore necessary to take into account all known causal conditions, and not to limit the inquiry to the study of bitemporal hemiopia due to any one lesion such as, for instance, pituitary tumour.

Three theories have been advanced to account for the field changes, namely: pressure, traction, and local intoxication. The reasons for and against each of these are discussed in considerable detail, the conclusion being that probably both toxic and mechanical causes are concerned, the latter mainly in the later stages.

The peculiar sequence of the field changes is explained by the way in which the crossed and uncrossed fibres are spread out in broad loops in the chiasma, a histological discovery which now receives confirmation from clinical symptoms. The atypical termination of some cases is ascribed to the increase in the size of the tumour, causing mechanical pressure effects to become predominant, thus "swamping" the more typical and regular changes. The author's conclusions, some of which are only tentative, are as follows:—

1. The perimetric defects in bitemporal hemiopia follow a typical or normal course of development. Commencing in the upper outer quadrant, the field is involved in a circular manner, the loss proceeding clockwise in the right field and counter-clockwise in the left, so that the upper nasal quadrant remains

longest. This is the typical course, and occurs in the majority of cases but, naturally, not in all.

2. The central defect or scotoma behaves in the same way, developing like a little field within the field.

3. This type of field defect is due to interference with the chiasmal fibres, but is largely independent of the exact nature of the ultimate cause; it occurs in bitemporal hemiopias from a variety of causes.

4. The immediate cause is very probably a chiasmal neuritis, a lesion comparable to that which, acting in the optic nerve, produces the symptoms of retrobulbar neuritis.

5. The cause of this chiasmal neuritis is not definitely known. In all probability it is set up in some way by pressure in many cases. The access to the chiasmal fibres of irritating toxic substances derived from the causal lesion may be the real cause; and the presence of these substances may in some cases indirectly arise from pressure. In some cases the chiasma may directly participate in an inflammatory process. The relation of the infundibulum to the chiasma is very probably of importance.

6. In tumour cases, and probably in some cases without tumour, mechanical pressure effects also act, mainly in the later stages, when the tumour has reached a relatively large size.

7. In the later stages the normal type of progress of the field changes may be "swamped" by mechanical pressure effects and greatly altered.

8. These observations provide evidence from the clinical side in support of the looped arrangement of the fibres in the chiasma.

9. They also indicate that the papillo-macular bundle forms a little chiasma within the chiasma, and that its fibres are similarly arranged.

The paper is illustrated by two coloured plates and twenty-eight figures in the text.

AUTHOR'S ABSTRACT.

THE MOST EFFICIENT ANAPHRODISIAC. W. J. ROBINSON, *Med.*
(408) *Record*, 1917, xci., p. 153.

ROBINSON recommends magnesium sulphate in doses of 20 to 40 grammes in half a glass of water once or twice a day.

Unlike potassium bromide, it does not cause any mental depression.

J. D. ROLLESTON.

THE DIAGNOSTIC VALUE OF SPINAL FLUID AND WASSER-

(409) **MANN TESTS IN PSYCHIATRY.** E. H. FELL, *Amer. Journ. Insan.*, 1917, July, No. 1, p. 41.

FIVE hundred cases have been subjected to a complete serological examination: 215 of these cases were luetic (paresis, tabo-paresis, and cerebro-spinal syphilis), and 285 were non-luetic.

Of the luetic cases, 27 had 6 or fewer cells per cm., while of the non-luetic group 10 cases—7 organic and 3 dementia præcox—showed a slight increase in cells.

In the luetic group, 7 cases did not show an increase of globulin, while in the non-luetic 16 had a globulin increase, 9 of these organic cases, 5 dementia præcox, 1 manic, and 1 unclassified.

In the luetic group the Wassermann reaction was positive with the blood serum in 91.6 per cent., and with the spinal fluid in 94 per cent. In the non-luetic group the spinal fluid never gave a positive reaction, but in 27 cases, 14 of which were dementia præcox, the blood serum gave a positive reaction.

D. K. HENDERSON.

PSYCHIATRIC ASPECTS OF PELLAGRA. WILLIAM C. SANDY, (410) *Amer. Journ. Insan.*, 1917, April, No. 4, p. 609.

DURING the latter half of the year 1915, 606 cases were admitted to the South Carolina Hospital for the Insane, of whom 160, or over 26 per cent., were pellagrins. Of these the largest number were white women, the others in reverse order of frequency being coloured women, coloured men, and white men. The most frequent ages among the males were from 20 to 70, among the females 20 to 50. During a period of twelve months 52 per cent. of the white males, over 51 per cent. of the white females, over 53 per cent. of the coloured males, and over 80 per cent. of the coloured females died from pellagra.

Out of a series of 160 cases the infective exhaustive psychosis, a condition characterised by delirium, confusion, disorientation, hallucinations, and physical exhaustion, occurred in 35 per cent. of the cases. In these cases the prognosis is grave, especially when accompanied by symptoms of cerebral irritation or central neuritis. The manic-depressive psychoses was seen in over 11 per cent., the depressed phase being more common than the manic.

The senile psychosis appeared in 10 per cent., dementia præcox or allied conditions in over 12 per cent., and 14 per cent. were left unclassified.

Rest in bed with careful nursing, and a proper well-balanced diet, appear to be the essential features of treatment.

D. K. HENDERSON.

FATIGUE AND ALCOHOL. A. F. STANLEY KENT, *Lancet*, 1917, ii., (411) July 28, p. 107.

THE taking of alcohol has little immediate effect upon the physical side of fatigue, whatever its ultimate result may be. The amount of output may perhaps be increased to a small extent for a time, but the increase soon falls off and is replaced by a diminution. The influence of alcohol upon the psychical side of fatigue is more profound. It leads immediately to a feeling of renewed vigour and increased strength. But here also the effect is transitory, and repeated doses must be taken if anything lasting is required.

Fatigue involves physical discomfort which can be diminished by drink, and as the effect is transitory the dose will be repeated often and industrial alcoholism established.

Alcoholism follows industry, but its severity is dependent on the conditions of industry. Conditions determine fatigue; fatigue induces men to drink. Clearly, therefore, if conditions are improved, fatigue and drinking will diminish.

The true cure for alcoholism is the provision of decent surroundings in the factory and in the home, adequate wages, leisure, and relaxation, clubs, recreation rooms, indoor and outdoor games—in short, the elements of a healthy, full, and interesting life in place of a mere existence without interest, without pleasure, and without hope.

A. NINIAN BRUCE.

PSYCHIATRY.

GENERAL PARALYSIS IN GENERAL HOSPITALS. (Paralysia
(412) *geral dos alienados nas enfermarias de Clinica Medica.*) O.
CLARK, *Brazil-Medico*, 1917, xxxi., p. 157.

SYPHILIS in Brazil is a milder disease than in cold countries, although only a small number of patients undergo specific treatment. General paralysis has recently shown a progressive increase, though it is far from being as frequent as it is in Hungary, which is the centre for general paralysis, as India is for plague, and Mexico for yellow fever. Thus, in the National Hospital at Rio, the percentage of general paralysis among all the insane is 6 per cent., as compared with 33 per cent. in Hungary (*cf. Review*, 1916, xiv., p. 571).

J. D. ROLLESTON.

BIOLOGIC ASPECTS OF DEMENTIA PRÆCOX. F. W. LANGDON,
(413) *Amer. Journ. Insan.*, 1917, April, p. 681.

THE author summarises the views of Kraepelin, Meyer, and Bleuler, and designates them as the "specific," the "sub-evolu-

tional," and the "psychogenic" hypotheses. In considering the sub-evolutional hypothesis great stress is laid, as evidences of atavism and for diagnostic purposes, on the "Kraepelinian handshake," and on the Simian type of hand as described by Stoddart.

D. K. HENDERSON.

**THE MANIC-DEPRESSIVE AND DEMENTIA PRÆCOX PSY-
(414) CHOSSES: THEIR DIFFERENTIAL SYMPTOMATOLOGY.**

H. H. DRYSDALE, *Amer. Journ. Insan.*, 1917, April, p. 627.

A FORMAL enumeration of the main symptoms met with in these two disorders.

D. K. HENDERSON.

**A PSYCHOSIS PRESENTING SCHIZOPHRENIC AND FREUDIAN
(415) MECHANISMS WITH SCHEMATIC CLEARNESS.** C. C.

WHOLEY, *Amer. Journ. Insan.*, 1917, April, p. 583.

AN excellent concise report of a case showing the value of analysis. The case does not lend itself to an abstract, and should be read in the original.

D. K. HENDERSON.

MANIC-DEPRESSIVE PSYCHOSIS IN THE NEGRO. E. M.

(416) GREEN, *Amer. Journ. Insan.*, 1917, April, No. 4, p. 619.

AN interesting paper which refutes the popular supposition that the manic-depressive psychosis is less frequent in the negro than in the white race. During the past six years 2,877 negroes were admitted to the Georgia State Sanitarium, of whom 501, or 17·4 per cent., were diagnosed as manic-depressive. The manic form is very much more frequent than the depressed, *e.g.*, out of 443 cases the phases exhibited were as follows:—

Mania -	-	-	-	-	325
Depression	-	-	-	-	62
Circular	-	-	-	-	26
Mixed -	-	-	-	-	23
Form undetermined	-	-	-	-	7

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D. K. HENDERSON.

THE EPHEBIC PSYCHOSIS. ARRAH B. EVARTS, *Amer. Journ. Insan.*,
(417) 1917, July, No. 1, p. 61.

FOUR very interesting cases are reported occurring in young coloured girls round the period of adolescence. In each of these

cases the psychosis remained at a fairly superficial level, and was much more in the nature of a reaction to the strain of adolescence than anything else. By ordering the patient's life, and by seeing that menstruation was firmly and satisfactorily established, a betterment was brought about ending in the patient's complete recovery. The term *ephebic*—from the Greek word *ephebos*, meaning youth—is introduced not with the idea of replacing the term *dementia præcox*, but simply as a means of drawing attention to the causative factors.

D. K. HENDERSON.

SOME OBSERVATIONS ON THE RELATIONSHIP BETWEEN
(418) **SYPHILIS OF THE NERVOUS SYSTEM AND PSY-**
CHOSSES. L. E. LOWREY, *Amer. Journ. Insan.*, 1917, July, No. 1,
p. 25.

THIS paper is based on an analysis of eleven cases showing divergence in the clinical and serological pictures. In this connection Southard and Solomon have reported a group of five cases showing the serology of general paresis or cerebro-spinal syphilis. Four of these cases were not considered insane, and the fifth case presented a typical manic-depressive attack, with recovery, plus positive serology. Barrett has reported a series of nine cases with manic-depressive symptomatology and course, and with the serology of neuro-syphilis.

The author reports four cases which he considers to be cases of *dementia præcox*, all of whom had serological findings similar to those found in general paralysis. Three of these cases seem to be conclusive enough, but the second case reported reminds one of a case of general paralysis on a hereditary basis. Two cases of constitutional inferiority with positive serological findings are also reported, and at autopsy both these cases showed the histological picture of general paralysis. These cases were originally diagnosed as cases of general paresis. Two of these have run an extremely long course, and one is now like a late case of *dementia præcox*, while the other has shown a negative Wassermann reaction in blood and fluid. The third case has been constantly hallucinated, and now shows little but these hallucinations.

D. K. HENDERSON.

TYPES OF DELINQUENT CAREERS. BERNARD GLUECK, *Mental*
(419) *Hygiene*, 1917, April, No. 2, p. 171.

In this paper Glueck, who is director of the psychiatric clinic at Sing Sing Prison, analyses fifty consecutive admissions. Twenty-eight, or 56 per cent., suffered from conditions capable of

affecting conduct seriously, and represent special problems, as follows:—

Syphilis of the central nervous system	-	4	per cent.
Mental defects plus syphilis	-	4	"
Mental defect	-	26	"
Alcoholism plus other grave affections	-	6	"
Morphine deterioration	-	2	"
Insane	-	14	"

Tables are shown giving the racial factors, hereditary factors, school attendance, onset of delinquent careers, and crimes of first offenders. Interesting and instructive case records setting forth the problem to be dealt with in the individual case are reported.

In the place of the present Sing Sing Prison there is to be built an institution which is to serve for the reception and classification of all prisoners of the State of New York, and opportunity is to be provided for the application of such scientific methods of procedure as may aid in administering the State's penal problem. Each prisoner will be detained in the reception building so long as may be necessary to definitely determine his physical and mental condition, and to decide what treatment, occupational or otherwise, will be most efficacious. The average prison population may be divided as follows:—

1. The normal prisoner who is capable of learning a trade.
2. The normal prisoner who is more or less advanced in years, whose occupation has been that of an unskilled labourer.
3. The insane delinquent.
4. The defective delinquent.
5. The psychopathic delinquent.

These patients, as best suits the case, will then be sent to the two industrial prisons of the State, or to the agricultural prison, or to the hospital for the criminal insane, or to an institution to be built for defective delinquents, or to either of the two latter institutions. The penal problem is held to be a psychiatric one.

D. K. HENDERSON.

SOME CRITERIA FOR THE EVALUATION OF MENTAL TESTS

(420) **AND TEST SERIES.** FLORENCE MATEER, *Mental Hygiene*, 1917, April, No. 2, p. 241.

AN excellent paper uttering a warning against the too-general and too-uncritical use of mental tests. In the first enthusiasm testing became largely a method of plus and minus, and "his

mental age is so much," all crime was feeble-mindedness, all immorality meant lack of mentality sufficient to control one's self. The psychological significance of the tests, controlling factors in the environment, and physical handicaps of the individual were largely ignored. Many of the so-called Binet testers could not have detected these, nor have evaluated them, even if they had wished to, because of the double lack of training and experience. There has been so much variation in test results reported by different examiners that nowadays attempts are being made to get a broader and more detailed standardisation of tests. The mental defective evidences his defects in more ways than one, and it is the sum of everything in the physical, mental, and environmental picture which constitutes and determines whether a child is normal or abnormal.

Mental tests individually and serially are not final. They are able to record the grosser quantitative differences between individuals, but comparatively little has been done on the standardisation of qualitative variations. We should recognise their shortcomings so that we may improve on them. "When all of the individual variations of the subject have been studied and evaluated, when sufficient data have been assembled to make a clinical picture of brain activity which is coherent and complete, then, and then only, is diagnosis allowable."

D. K. HENDERSON.

SURVEY OF MENTAL DISORDERS IN NASSAU COUNTY, NEW YORK, JULY-OCTOBER 1916. *Psychiatric Bulletin*, 1917, April, No. 2, p. 109.

THIS survey, comprising 122 pages, is too important and too detailed to lend itself to an abstract. The work was carried out under the direction of Dr A. J. Rosanoff, with whom were associated a staff of four physicians, a psychologist, and fifteen field workers. One cannot do better than quote from the director's summary.

The principal question raised is not, What is the percentage of "insane," or "feeble-minded," or "mentally defective" persons in the population? But rather, What instances of social maladjustment, sufficiently marked to have become the concern of public authorities, are, upon investigation, to be attributed mainly, or in large measure, to mental disorders?

Thus the main object of the survey was to study the nature of the relationship between social maladjustments and mental disorders. Accordingly, two independent systems of classification were adopted—a medical and a sociological one.

This plan has brought to light, on the one hand, cases clearly abnormal from a scientific standpoint, yet without marked social maladjustment, and, on the other hand, cases of marked and persistent social maladjustment clearly connected with psychic anomalies, yet not assignable to any of the groups in the medical classification, excepting, of course, that of "disorders of uncertain nature or etiology."

Four special districts, with a total population of 4,668, were selected for intensive investigation, *i.e.*, a house to house canvass.

Thus it was hoped to secure a certain amount of control material to serve for the evaluation of the method employed in the county at large, and to furnish an idea of normal social standards prevailing in the county. The cases investigated were classified in four groups:—(1) 4,129 normal cases with no "abnormal" relatives; (2) 2,732 normal cases, but with "abnormal" relatives; (3) 583 cases of "doubtful" mentality; (4) 1,592 "abnormal" cases.

In group 1 the native of native parentage show 0.3 per cent. of illiteracy; the native of foreign or mixed parentage show 0.4 per cent.; while the foreign born show 17.3 per cent.—a contrast due to unequal educational opportunity.

In group 4 the native of native parentage show 18.8 per cent. illiteracy, and the foreign born show 23.6—a showing due, in the case of the native born, to low degree of educability, and, in the case of the foreign born, to low degree of educability and poor educational opportunity.

The 1,592 "abnormal" cases were grouped, medically, as follows:—Insane, 24.5 per cent.; feeble-minded, 39.8 per cent.; epilepsy, 4.5 per cent.; other groups (Huntington's chorea, cretinism, &c.), 31.2 per cent. Sociologically, these cases were grouped as follows:—Retardation in school work, 10.6 per cent.; truancy, unruliness, &c., 1.3 per cent.; sex immorality, 7.3 per cent.; criminal tendency, 5.0 per cent.; vagrancy, 0.1 per cent.; dependency, 17.6 per cent.; inebriety, 20.1 per cent.; drug habits, 0.3 per cent.; domestic maladjustment, 0.9 per cent.; medical cases, 3.6 per cent.; other groups, 23.1 per cent.; no maladjustment, 10.2 per cent.

Cases of dependency, of inebriety, of sex immorality, and of criminal tendency constitute a mass of psychiatric material requiring consideration as urgently as any group of "insane."

Of 946 cases judged to require institutional treatment, only 365 were found in institutions, the remaining 581 being at large.

The work in the schools was confined to eight school districts, where 2,500 children were examined by medical officers of the United States Public Health Service. Among these, 51 were

judged to be "abnormal" (feeble-minded, psychopathic personality, epileptic), 14 probably feeble-minded, and 116 retarded.

A number of interesting case-records are given, the whole work is discussed, and a model psychiatric system is proposed.

D. K. HENDERSON.

DECLINE OF ALCOHOL AS A CAUSE OF INSANITY. H. M. (422) POLLOCK, *Psychiatric Bulletin*, 1917, April, No. 2, p. 103.

DURING the eight fiscal years from 1st October 1908 to 30th June 1916, 58,011 patients were admitted into the New York State Hospitals. An inquiry into the habits of all these patients with respect to the use of alcohol was made at the time of admission, and a statistical data card was prepared for each patient, and forwarded to the Bureau of Statistics. Among the alcoholic first admissions the males constitute approximately three-fourths, and the females one-fourth of the total; among the alcoholic re-admissions the males constitute about four-fifths, and the females one-fifth of the total number. In 1909 the percentage of alcoholic cases among all admissions was 10.1, but in 1916 this had become reduced to 5.7.

In 1909 the percentage reported as using alcohol to excess was 28.7, while in 1916 it was 18.5.

"The evidence herein set forth of the general decline of alcohol as a cause of insanity in New York State seems conclusive. The alcoholic cases annually admitted to our hospital since 1909 have decreased both relatively and absolutely, and intemperance as a contributing factor in causing mental disease has gradually diminished.

"It is probable that these results are due principally to improvement in the habits of the general population with respect to the use of alcohol."

D. K. HENDERSON.

THE RECEPTION, EXAMINATION, AND CARE OF NEW
(423) **ADMISSIONS.** CHARLES G. WAGNER, *Amer. Journ. Insan.*, 1917, April, No. 4, p. 673.

A DETAILED account of the reception and management and treatment of patients received into the New York State Hospitals. Every institution for the insane should possess a reception building designed and equipped especially for cases of the acute class. This reception building should have single rooms, small dormitories for the suicidal cases, and general wards and sitting-rooms comfortably and attractively furnished. The importance of hydrotherapy, occupation, games, good food, and careful nursing are all dwelt upon.

D. K. HENDERSON.

PSYCHONEUROSES, PSYCHOSES, AND MENTAL DEFICIENCY
 (424) **IN 2,000 CASES, CONSIDERED ESPECIALLY FROM THE**
STANDPOINT OF ETIOLOGICAL INCIDENTS AND SEX.

ALFRED GORDON, *Amer. Journ. Insan.*, 1917, April, No. 4, p. 721.

THIS report is based on 1,100 cases of psychoneuroses, 660 psychoses, and 240 cases of mental deficiency.

In 575 cases of the psychoneuroses disappointments of various sorts occurring against all expectations were the causative factors, while the next most common cause was loss of affection of children for their parents (250 cases). Of the 1,100 cases 813 were women, and there were more unmarried than married ones.

In the largest majority of the cases of manic-depressive insanity incidents of a depressive character preceded the onset of the psychosis, but of 150 cases of dementia præcox factors of an exhausting character, *e.g.*, preparation for examinations, were found in fifty-six instances.

Thirty-nine out of fifty patients suffering from involutional melancholia had a history of a morbid heredity, or else individual features of a psychoneurotic type. The incidental factors preceding the onset were of a strongly emotional and depressive character.

In twenty-five cases of acute confusion with delirium there was a direct relationship between the preceding etiological factor, *e.g.*, typhoid, pneumonia, trauma, and the disorder. Out of 240 cases of mental deficiency 180 showed psychasthenic symptoms, and in these "sudden fright" was practically the only exciting cause. In the remaining sixty cases which showed symptoms of a psychosis, alcohol, excessive cigarette-smoking, and masturbation were the three principal causative factors.

D. K. HENDERSON.

MADNESS AND UNSOUNDNESS OF MIND. CHARLES A. MERCIER,
 (425) *Journ. Ment. Sci.*, 1917, Oct., p. 488.

MADNESS and unsoundness of mind are not the same thing; madness includes more than unsoundness of mind, and unsoundness of mind very often occurs in the sane (*e.g.*, tinnitus), and is, indeed, one of the most frequent disorders of the sane.

It is not proved, and it is not provable, but it may be that in every case of madness there is disorder or disease of mind, or unsoundness of mind; but madness is certainly not the same thing as disorder, or disease, or unsoundness of mind, for if it were we could never observe it, for certainly we cannot observe what is going on in the minds of other people.

On the other hand, it is provable and it is proved that in every

case of madness there is disorder of conduct. When we certify a person as mad, we have not observed disorder of brain or disorder of mind; what we have observed is something the patient has said or something the patient has done; and what the patient says or does is not part of his brain or part of his mind; it is part of his conduct. If there is no disorder, or failure, or defect, or fault in anything he says or does, it does not in the least matter what the state of his brain is, or what the state of his mind is—we cannot, for example, see or hear the delusion in another person's mind; all we can see or hear is the expression of it in speech or gesture, or some other mode of conduct.

H. DE M. ALEXANDER.

THE ORIENTATION OF HUMAN AND ANIMAL FIGURES IN
(426) **ART.** J. BARFIELD ADAMS, *Journ. Ment. Sci.*, 1917, Oct., p. 506.

Mlle. V. KIPIANI (quoted by Mlle. Joséfa Ioteyko) asserted all children orient to the left all pictures they are asked to draw, and all that is oriented by the hand of man (as exemplified in the original pictures of numerous painters, as well as the reproductions of the pictures from all the picture galleries of Europe) looks, walks, runs, and flies towards the left on canvas or paper.

Mlle. V. Kipiani states that it is so because we design with the right hand only. The ancient Greeks, Egyptians, and the modern Chinese and Japanese orient by preference to the right, on account of their centripetal handwriting: and among the Chinese and Japanese the right side of the picture contains the principal idea, whereas it is the left side of the picture among Europeans where the principal scene unrolls itself.

The author, by a series of observations among children, an examination of the pictures in the galleries of England and the Continent, and a study of the remains of the decorative and plastic arts practised by the ancient Greeks, Egyptians, and other antique peoples, is satisfied that this statement is incorrect and asserts that the idea of form is received principally through the aid of the oculomotor nerves, thus bringing both cerebral hemispheres into action.

“Artists orient the figures in their pictures according to the necessities of the picture, and not in obedience to some unconscious impulse which compels the almost constant orientation of animate and inanimate objects from right to left.”

H. DE M. ALEXANDER.

THE CARE AND TREATMENT OF THE INSANE IN THE
(427) **COUNTY INSTITUTIONS OF PENNSYLVANIA.** WILLIAM
C. SANDY, *Mental Hygiene*, 1917, April, No. 2, p. 279.

IN 1914 Haviland made a survey of the institutions in Pennsylvania caring for the insane, and revealed a state of amazing neglect. He found the insane cared for in county asylums under the most distressing conditions, and the State system of hospitals so conducted as to be inefficient. Attempts were made to secure proper legislation to deal with this state of affairs, but failed. An effort is now again being made this year, and this article is a summary of a survey made by Sandy. Sandy practically restates the condition of affairs reported by Haviland, and shows how the majority of the institutions are insufficiently equipped in personnel, in fire protection, toilet and bathing facilities, provisions for examination, treatment, classification, occupation, recreation, and the like. The majority of the county institutions are under lay supervision, a political appointee, whose retention in office is subject to the whims of Poor Directors. The care is largely custodial, and treatment is not attempted.

D. K. HENDERSON.

HOW MAY WE DISCOVER THE CHILDREN WHO NEED
(428) **SPECIAL CARE?** ROBERT M. YERKES, *Mental Hygiene*, 1917,
April, No. 2, p. 252.

THE first ten years of life is by far the most important, and during that period children should be classified into one of the following groups: (1) the intellectually superior, or super-normal; (2) the intellectually inferior, or subnormal; (3) the intellectually dependent; (4) the affectively or instinctively defective; (5) the mentally normal, typical, or average.

Humanitarian work with the feeble-minded is of the utmost importance, but it is even more important that careful attention and special care be given to the intellectually superior. The plan proposed for picking out the children to be treated and helped is as follows:—A staff of well-trained and experienced experts, including a physician, a psychologist, an educator, and a social worker, should be organised. These experts should study the entire school population of some city, county, or State by means of the best methods of physical and mental measurement available. The children should be examined in groups of twenty to fifty for the more gross defects, and then those individuals needing a more detailed examination (usually 10 to 15 per cent.) would be re-examined. A detailed report with suggestions or

definite recommendations concerning proper, desirable, or urgent educational, medical, or vocational treatment would then be given.

D. K. HENDERSON.

DREAMS AND THEIR INTERPRETATION. ROBERT ARMSTRONG
(429) *Jones, Amer. Journ. Insan.*, 1917, April, No. 4, p. 655.

IN this article the author gives an account of the history of dreams and of the explanations, from time to time, advanced to account for them. He feels that the ideas of Freud have been greatly over-emphasised, and describes those "Freudians who urge sex as the basic origin of all dreams, of all obsessions, and of all longings, impulses and neuroses as sex-intoxicated." Among the closing statements is the following: "It is a comfort to know that we dream most about events to which no attention has been paid."

D. K. HENDERSON.

RECENT TRENDS IN PSYCHIATRY. CHARLES G. WAGNER, *Amer.*
(430) *Journ. Insan.*, 1917, July, No. 1, p. 1.

THE presidential address at the seventy-third meeting of the American Medico-Psychological Association. The author briefly recounts recent advances in psychiatric work, and reserves a few paragraphs in which to express his disapproval of psychoanalysis and of those who use such a method.

D. K. HENDERSON.

OBSERVATIONS ON CRANIAL ASYMMETRY. H. M. ADLER,
(431) *Amer. Journ. Insan.*, 1917, July, No. 1, p. 89.

THE dividing line between normal and abnormal asymmetry is not clean cut, but in this paper the author discusses extreme cases. The cases considered are those which have shown a difference in height of the two eyes of over 3 mm., compared to a perpendicular drawn through the median plane of the skull.

The slope of the head was taken by means of a "conformateur" such as is used by hatters in fitting men's hats, and a series of outlines are illustrated. These outlines show a flattening and receding of the skull on the retarded side, and also a noteworthy prominence in the parietal region, usually on the right side. This corresponds to right-handedness, and in cases where a person is not right-handed this prominence is absent. In left-handedness little difference is found between the two parietal regions. Out of 1,000 cases examined, 719 were found with marked asymmetries,

while of 86 employees only 5 were found to be markedly asymmetric.

Asymmetry of the cranial vault is thought to be due, not to premature closure of the sutures, but rather to an arrest of development of the soft parts within, and it is thought to indicate a tendency to psychopathy.

D. K. HENDERSON.

THE CRIMINAL INSANE AND INSANE CRIMINALS. PAUL E. (432) BOWERS, *Amer. Journ. Insan.*, 1917, July, No. 1, p. 77.

EPILEPSY, paranoia, and feeble-mindedness are the three most important forms of mental alienation which often lead directly to crime. The author emphasises the differences between the medical and legal view point in regard to the treatment of insane criminals, and shows how justice, not knowing all the medical facts, may go far astray. The percentage of recoveries in insane criminals is very much smaller than in ordinary hospitals for the insane.

D. K. HENDERSON.

THE NEED OF CLOSER RELATIONSHIP BETWEEN PSYCHIATRY AND THE MEDICAL SCHOOLS. A. H. RUGGLES, *Amer. Journ. Insan.*, 1917, Oct., No. 2, p. 149.

It is necessary to encourage well-trained medical students to enter psychiatry, to raise the standards of psychiatric teaching in this country, to stimulate a greater co-operation between those working in mental medicine, and to help in a wider dissemination of psychiatric knowledge.

D. K. HENDERSON.

SUBSIDISING MENTAL DEFICIENCY—THE "PAUPER IDIOT ACT" OF KENTUCKY. T. A. HAINES, *Mental Hygiene*, 1917, April, No. 2, p. 274.

AN historical document with a criticism of present methods of care for the feeble-minded in Kentucky. At the present time an allowance of £15 per annum is paid from the State treasury to the committee for each pauper idiot, a state of affairs which puts a premium upon having feeble-minded children, and by pensioning them favours further multiplication.

D. K. HENDERSON.

Reviews

INSTINCT IN MAN. A Contribution to the Psychology of Education.
(435) JAMES DREVER, M.A., B.Sc., D.Phil. Pp. x and 281. Cambridge University Press.

THE science of psychology owes a great deal to the work of medical men; yet it is true that the medical student is still insufficiently trained in a science which obviously concerns his work very nearly, and which might be of the greatest practical use to him. Every successful physician owes much of his success to what we may call his working knowledge of psychology; but if he understood the theory which lies behind his practice he would walk more surely and attain to even greater success. For the alienist—the man who would minister to a mind diseased—a thorough knowledge of psychology should surely be regarded as indispensable. It is now widely recognised that emotional disturbances in particular play an enormously important part in the psycho-neuroses; hence any psychological work which throws light on the laws of our emotional nature should be assured of a welcome from the psychiatrist.

From this practical point of view the most illuminating and helpful book that has appeared of recent years is McDougall's "Introduction to Social Psychology." In this work McDougall seeks out the springs of human action, which he finds in certain primary instincts, each characterised by a distinctive emotional experience (*e.g.*, fear) and a specific impulse. By the intermingling of these instincts complex emotional experience is attained, and by the help of memory and habit character is formed. The foundation of every character is to be found in the native instinctive endowment, individual differences being largely accounted for by differences in the original strength of each of the primary instincts. If any one of these becomes hypertrophied or atrophied, abnormalities of character result.

The first part of the book before us consists of an interesting and valuable historical account of the place and meaning of instinct in modern times; the second and more important part may be broadly regarded as a critical examination of McDougall's theory of human nature. The writer finds himself in essential agreement with McDougall, differing from him only in points of analysis, the importance of which will appear more to the trained psychologist than to the practical man. His general classification of the inborn tendencies agrees on the whole with that of McDougall, while his treatment of the emotional factor in these

tendencies is, we consider, a definite step forward in our understanding of this extremely obscure region of our mental life.

According to Dr Drever, the affective aspect of the instinct is to be found in interest, specific emotions resulting only when there is tension or blocking in the activity prompted by the instinct. "Biologically the function of emotion is apparently to reinforce impulse and interest. This reinforcement will be necessary in two cases, either where an obstacle must be surmounted, or where a more or less prolonged course of trying to find the appropriate reaction is necessary, owing to the fact that no neural prearrangement provides for the precise action in a particular case. In the first set of circumstances, in addition to the appropriate emotion, whatever that may be, anger generally develops as a further reinforcement. In the second, anger will not meet the needs of the situation, since only actions of a certain kind will satisfy the impulse and interest involved, and only the appropriate emotion can secure such actions." It is thus the complexity of man's life, rendering it more and more difficult for inherited adjustments bringing about specific reactions to be available for every emergency that has brought with it the development of the large group of emotional instincts.

Working with an intentionally wide definition, Dr Drever is able to include as instincts certain innate tendencies already marked out as instincts by James, but for which McDougall was unable to find a satisfactory place owing to his more circumscribed definition. Dr Drever's concise classification of all man's innate tendencies, grouped under the headings Appetite Tendencies and Instinct Tendencies, seems to us both helpful and suggestive (p. 169).

Dr Drever then proceeds to discuss in detail the various innate tendencies. Dealing first with the specific instinct tendencies, he takes up in turn fear, anger, and the hunting instinct, the gregarious instinct, the acquisitive tendency, courtship and the self-tendencies, the parental instinct, and curiosity. Next follows a chapter on interests and sentiments, which, while agreeing in the main with McDougall's theory of the formation of character, yet throws valuable additional light on the process, and is full of suggestion for the interpretation of character both normal and abnormal. The general instinct tendencies, including play, experimentation, imitation, sympathy, and suggestibility, are next dealt with, and a short concluding chapter is devoted to the appetite tendencies.

Considering the position in psychological literature that has been accorded to the "Elements of Social Psychology," it is perhaps natural that Dr Drever is at times inclined to over-elaborate the differences between his own views and those of McDougall, and

in so doing he sometimes fails to render himself perfectly clear (*e.g.*, in his treatment of the "joy" emotions). In place of some of these criticisms we should gladly have welcomed a fuller exposition of the educational applications that Dr Drever finds in his psychological theory. Of these applications enough is given to whet our appetite for more. In his preface Dr Drever indicates that this constructive work may be forthcoming on some future occasion. The students of the present volume will look forward to that future occasion with eager anticipation.

MARGARET DRUMMOND.

BREEDING AND THE MENDELIAN DISCOVERY. By A. D. (436) DARBISHIRE, M.A. Cassell & Company, Ltd. London, 1911.

AN INTRODUCTION TO A BIOLOGY AND OTHER PAPERS. (437) By A. D. DARBISHIRE. Cassell & Company, Ltd. London, 1917. Pr. 7s. 6d. net.

THE first of the above works by the late Mr Darbishire should be read in connection with the second. The former gives an excellent exposition of Mendelism, although in some respects immature. Mutation is well illustrated and described, and an interesting account of Goss's experiments in plant crossing in 1820 is given, which, although inconclusive, are remarkably similar to Mendel's.

In the second book Darbishire had been influenced by Bergson's work, and his insight and critical powers are much quickened. The second work especially can be read with great profit by all interested in the mechanism of evolution.

The circumstances of Mr Darbishire's death lend additional interest to the reader.

PSYCHOLOGICAL MEDICINE. A manual on mental diseases for (438) **practitioners and students.** MAURICE CRAIG, M.A., M.D., F.R.C.P. Third Edition. Pp. xii+484, with 27 plates, some in colour. J. & A. Churchill, London. 1917. Pr. 15s. net.

DR CRAIG's manual on mental diseases is so well known that further recommendation is not required. In this edition it has been brought up to date and somewhat enlarged. This has necessitated a new chapter upon the psycho-neuroses occurring in men exposed to shell shock and strain of war. The writer has not been impressed by the results he has seen from psycho-analysis in war neuroses. Psycho-analysis has been more fully described, and the Mental Deficiency Act is explained.

Special reference is made to the fact that "there are still no facilities for the treatment of poor persons who are showing signs of nervous exhaustion; these continue to be allowed to drift on until many of them can be certified as insane." "For years we

have been waiting, and we still wait, the day when special hospitals will be founded and equipped with all that is necessary for the treatment of neurasthenic and fatigue states." Most useful information is given in the chapters upon moral imbecility, feigned insanity, and the relationship of insanity with law. The need for detailed study in the differentiation of organic from functional symptoms is strongly emphasised, as well as the importance of regarding mental disorders in the same light as physical illness.

This edition should undoubtedly maintain the reputation gained by the earlier editions.

TEXT BOOK OF OPHTHALMOLOGY. ERNEST FUCHS. Authorised (439) translation from the twelfth German edition; completely revised and reset, with numerous additions specially supplied by the author, and otherwise much enlarged, by Alexander Duane, M.D. Fifth Edition. With 462 illustrations. Pp. xxv+1067. J. B. Lippincott Company, Philadelphia and London. Pr. 30s. net.

THE present edition is stated to be in several senses a new work, as it contains a great many alterations made by the translator, and many additions and changes supplied by the author himself. The numerous pages of remarks in small print which were collected in earlier editions, as an appendix at the end of chapters, the value and interest of which were somewhat obscured by this arrangement, have been divided into shorter sections, each placed in close position to the part of the text to which it is related, and many of the more important items have been incorporated in the text. The section on the diagnosis of ocular paralyses has been rewritten, while the chapters on glaucoma, diseases of the retina and disturbances of motility, and the sections on refraction and accommodation, have been rearranged and added to. Among the many additions scattered through the book the following are to be noted: tuberculin and vaccine therapy, the visual field and colour testing, the mapping of scotomata and the blind spot, the etiology of trachoma, sclerosis of the cornea and of the choroid, the different forms of retinal degeneration, the varieties of accommodative troubles other than paralysis, &c.

This edition undoubtedly will take a high place amongst textbooks of ophthalmology, and presents in a singularly lucid way the general physiology of the eye, and the pathology, etiology, symptomatology, and treatment of eye diseases as a whole. Six chapters are devoted to operations, which are described with great care. The illustrations, many of which are coloured, all through the book are excellent. References to literature are not given. There is a large and very complete index.

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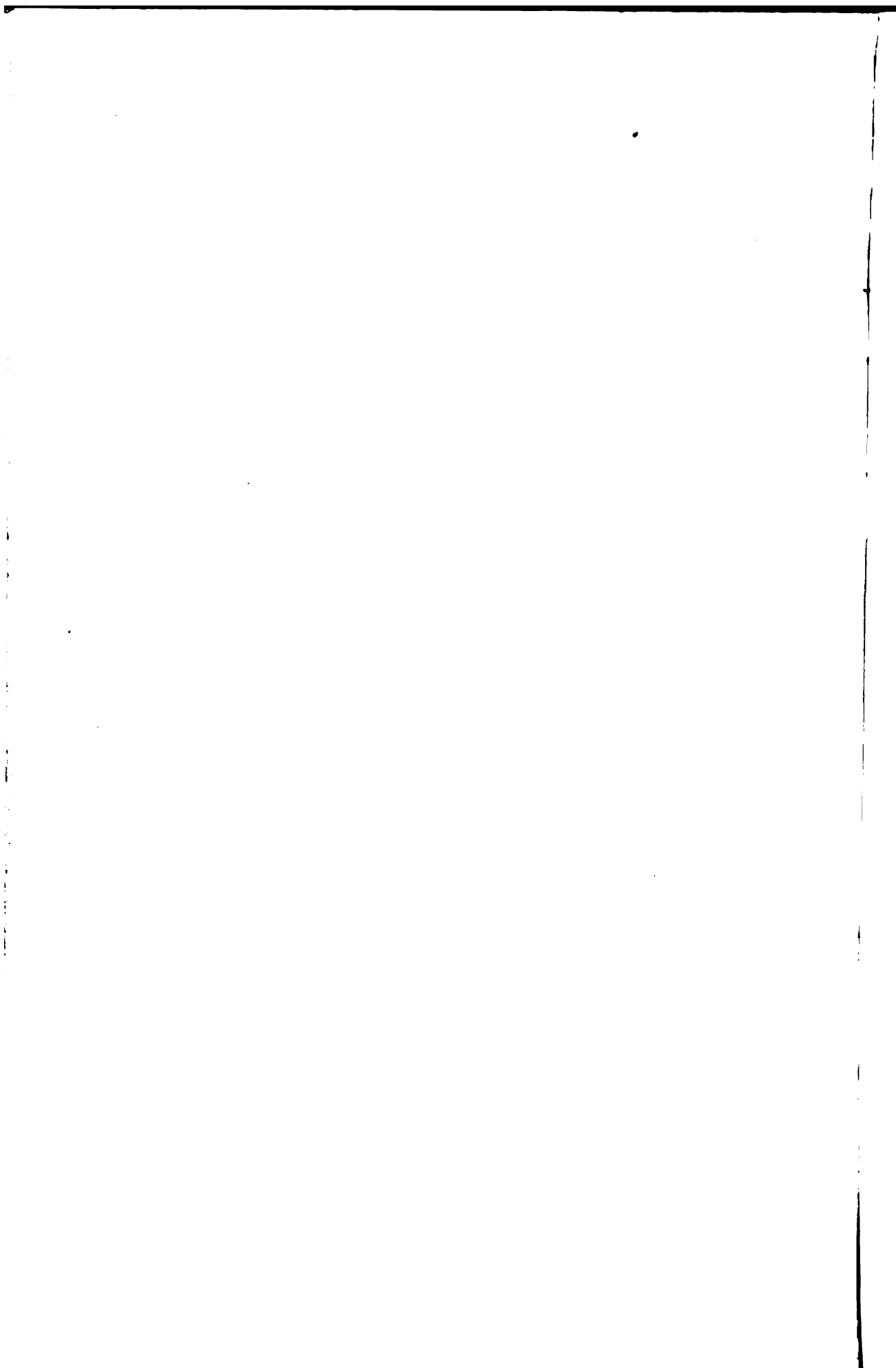
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